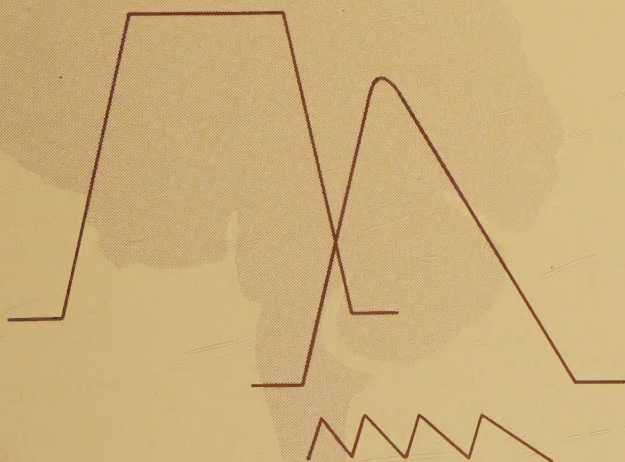


SACCADES, FAST PHASES OF NYSTAGMUS AND TRACKING EYE MOVEMENTS IN MAN

An electro-oculographic study

By
Lucyna Schalén



Lund 1981

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av

Lucyna Schalén

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Abstract An analysis of saccades, fast phases of nystagmus and tracking eye movements was made in normal subjects and in patients with defined lesions in order to examine the vulnerability of these eye movements. Peak velocity of saccades and fast phases of nystagmus and accuracy of saccades were analysed. In normal subjects saccades were faster than fast phases of nystagmus. Decreased peak velocities were found in brain stem lesions. In frontal lobe lesions and in vestibular lesions peak velocities were normal whereas in meningo-encephalitis increased peak velocities were observed. The accuracy of saccades was decreased in all types of lesions except the vestibular disorders. Tracking eye movements were analysed in terms of maximum velocity gain of smooth pursuit, number and amplitude of superimposed saccades, and square waves and the magnitude of excursion of the eye in relation to the target. Impairment of tracking showed a stereotyped pattern in disease: decrease in maximum velocity gain of smooth pursuit and increase in number of superimposed saccades. Although this pattern was present in different types of lesions, it was especially evident in cerebellar and brain stem disorders. An analysis of saccades, fast phases of nystagmus and tracking eye movements enhances our diagnostic skills and should be incorporated in routine otoneurological examination.			
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Date August 14, 1981

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**By
Lucyna Schalén**

**Department of Otolaryngology, University Hospital,
Lund, Sweden**

Lund 1981

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- I. Henriksson, N.G., Pyykkö, I., Schalén, L. & Wennmo, C. 1980. Velocity patterns of rapid eye movements. *Acta Otolaryngologica* (Stockholm) 89: 504-512.
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- III. Schalén, L. 1980. Quantification of tracking eye movements in normal subjects. *Acta Otolaryngologica* (Stockholm) 90: 404-413.
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Introduction

DEFINITIONS

Nystagmus is an oculomotor reflex consisting of a series of alternating slow and fast eye movements. Depending on the stimulus, vestibular or optokinetic nystagmus (OKN) can be distinguished. Interaction of vestibular and visual stimuli results in optovestibular nystagmus, which is the most common type of nystagmus occurring under natural circumstances (53, 142).

Saccades are rapid eye movements transferring the gaze between two points of fixation. They can be executed intentionally but also reflexively to visual, auditory, tactile or imaginary targets (24, 112).

Smooth pursuits are slow eye movements executed during fixation on moving visual objects. Efforts to perform smooth pursuits without a moving target usually produce small saccades. Occasionally, however, smooth pursuits can be recorded in trained subjects in darkness without visual fixation (33, 55).

Tracking eye movements are distinguished from smooth pursuits by consisting of both smooth pursuits and superimposed saccades. In natural circumstances the saccades are essential components of tracking, assisting the foveation (44).

Saccades and tracking eye movements in natural circumstances can be performed in horizontal, vertical or oblique planes. Nystagmus can be induced also in rotatory manner (53). In the present study only horizon-

tal eye movements were considered.

PHYLOGENETIC ASPECTS OF OCULOMOTOR SYSTEM

The various types of eye movements outlined above were first distinguished only at the beginning of the present century (37). Not until 50 years later was evidence published that horizontal saccades and smooth pursuits are controlled by two independently operating neuronal subsystems (106, 109, 110, 139, 140). Since then these two types of eye movement have been extensively studied in different species, revealing wide variability of the oculomotor controls, depending on the stage of phylogenesis (50).

The mechanism of the triggering of saccades and smooth pursuits is not known. However, on the basis of comparative phylogenetic considerations it was anticipated that voluntary eye movements are mediated by pathways partly separate from those involved in processing nystagmus (111). It also became evident that saccades, scanning the environment independently of the movement of the head, can be produced only in higher vertebrates whereas smooth pursuit eye movements are allegedly independent of head movements only in man and monkey (24, 53, 112).

Since the organization of the saccadic and smooth pursuit systems seemed to be analogous in monkey and in man (46) the monkey has been used as a study model for the eye movement system. However, as there are evident differences between the two species with respect to performance of the psychomotor tasks demanding motivation (30) and attention (24), conclusions regarding the human oculomotor system will require studies on humans in addition to animal experiments.

Animal experiments, however, provide important infor-

mation on organization of the oculomotor system at different sites e.g. sensory receptors (102), cortex (21, 67), cerebellum (71), brain stem (52) and external eye muscles (1). An understanding of these mechanisms may help us to interpret the disturbances of eye movements in patients with lesions in the central nervous system and may improve our diagnostic prospects. Therefore short review of studies on neuronal mechanisms of nystagmus, saccades and tracking eye movements will be presented.

VESTIBULAR NYSTAGMUS

Current knowledge on the organization of the vestibular system has been presented in recent comprehensive reviews (53, 142).

Influenced by the vestibular system, eye movements are induced to compensate for movements of the head. Head movement causes a flow of endolymph within the semi-circular canals, causing deviation of the cupula and triggering deviation of the eyes (38, 128). The basic elements of the vestibulo-ocular reflex arch are hair cells in the cupula, the afferent neurons of Scarpa's ganglion, interneurons within the vestibular nuclei and effector neurons in the oculo-motor nuclei (86).

It should be emphasized that not all afferent impulses to the oculo-motor nuclei are conveyed via the three-neuronal vestibular reflex arch (24, 130). Some of the impulses reach the oculo-motor nuclei via other multi-synaptic pathways in the brain stem. Thus, the brain stem is closely involved in controlling the vestibulo-ocular reflex arch (73), participating in enhancing or suppressing the vestibular responses (57). Therefore a lesion in any part of the vestibular projections - either within the three-neuronal reflex arch or within non-specified brain stem pathways - can cause a patho-

logical nystagmus.

Triggering of the fast phases of nystagmus seems to occur by activating the neuronal population of the paramedian pontine reticular formation (PPRF) as revealed by single unit recordings (73). Stimulation (73) and lesion studies (25) have indicated the importance of this structure for control of the fast phase of nystagmus. Distinct populations of cells in vestibular nuclei (122) and in reticular formation (90) may also be involved in generating the fast phases of nystagmus. Lesions at various sites in the brain stem are known to disturb the fast phase of nystagmus (25).

SACCADES AND TRACKING EYE MOVEMENTS

Stimulation (113) and ablation studies (82) have indicated the importance of the frontal eye field (Brodmann's area 8) for saccade generation. Single unit recordings in the monkey confirmed connection of the saccades with neuronal activity within the frontal eye field (22, 93). Recordings of presaccadic activity do not provide consistent results. Thus, no presaccadic activity is seen in the frontal eye field in monkey (22, 93), while in human studies on electrical brain activity (EEG) ramp-like potentials within the area of the frontal eye field preceded the onset of voluntary saccades (17, 80). The projection of impulses from frontal eye field terminates in contralateral brain stem nuclei, as shown by neuro-anatomical studies (5).

Single unit recordings from the posterior parietal cortex (Brodmann's area 7) in monkey indicate presaccadic activity in this region, though only in relation to saccades directed to targets of special interest in experimental animals (69, 84, 87).

There is, to the author's knowledge, no report giving evidence of cortical activity prior to smooth pursuit, though neurons active during smooth pursuits are found within the occipital (100, 101), the frontal (21), and parietal cortex (84, 87).

Extensive lesions in the occipital lobe can produce defective vision, with impairment of gaze. Accordingly, ablation and stimulation studies on the occipital cortex are difficult to interpret, as they may merely reflect visual defects.

On the basis of eye motor disturbances after unilateral ablation of the occipital cortex in monkey it was suggested that each occipital lobe may initiate both ipsi- and contralateral eye movements and that the smooth pursuit pathway terminates mainly in the contralateral brain stem (48). In contrast, the neuro-anatomical studies in monkey (23) and clinical observations (64, 120, 132) indicate that smooth pursuit pathway projects to the brain stem and terminates ipsilaterally.

As mentioned earlier, testing of saccades and smooth pursuits in man requires volition whereas in animals, including primates, saccades and smooth pursuits are performed only after conditioning of the animals. Consequently, the results of animal experiments do not allow direct conclusions regarding the mechanisms triggering saccades and smooth pursuits in man. Parallel studies in man would appear necessary.

PROCESSING OF VISUAL SIGNALS

The visual information prompting execution of eye movements is processed through the striate visual system and also via extra-striate pathways. Apparently, disturbance of the sensory input at any site of visual

projection can disturb the oculomotor output. Thus, some basic knowledge of the organization of the visual part of the oculomotor system should be presented. As yet, however, the oculomotor projections of the visual system are not known in detail.

The striate visual pathway consists of several neurons; afferent neurons synapsing in the retina and in the lateral geniculate body (LGB) and terminating in the primary visual cortex (area striata). The first recognition of velocity and direction of the signal takes place already in the retina (18, 83, 102). The primary visual cortex (Brodman's area 17) and associative visual cortex (Brodman's areas 18 and 19), however, seem to be involved in the more precise recognition of velocity and position (66, 67, 94, 101, 143). The visual cortical areas exhibit activity in relation to saccades (39, 66, 75, 101, 129) and smooth pursuits (67, 100, 101).

The exact pathway of the visual oculomotor projections to the brain stem and cerebellum is not known; however, it has been demonstrated that visual information is relayed in pretectal nuclei (26, 104) and further channelled via the inferior olive to the flocculus (70). Since direct visual projections to the vestibular nuclei have not been demonstrated, the cerebellum is the only well documented site of vestibular and visual interactions. However, it is likely that extracerebellar visual projections to the vestibular nuclei do exist, since visual influence on vestibular neurons is not lost after cerebellar ablation (74).

The extra-striate visual pathways terminate in the brain stem. One accessory optic tract (AOT) (88) leaves the visual pathway just posterior to the chiasma opticum and - omitting the LGB - projects to the upper brain stem. Projections of the AOT reach the individual Purkinje cells in the cerebellum as climbing fibres (125) via the dorsal cap of the inferior olive (136) or as mossy fibres via direct projection to the flocculus (89). The exact role of the AOT in the oculomotor functions is still not clear. However, results of single unit recordings in monkey (98) and rabbit (125) indicate that AOT may provide the cerebellum with visual information necessary for control of reflexive and voluntary eye movements.

PONTOCEREBELLAR PROCESSING OF EYE MOTOR IMPULSES

According to Ito (71) the cerebellum controls the brain stem structures. Certain populations of cerebellar neurons exhibit activity modulation, depending on the position of the eye in the orbit and while per-

forming rapid eye movements (97) or responding to eye velocity during smooth pursuit (85). Ablation studies (4, 108) also indicate that the cerebellum is necessary for execution of saccades and smooth pursuits.

The brain stem, especially the paramedian pontine reticular formation (PPRF), in the monkey, contains neuronal elements which participate in executing of all types of eye movement: phasic units active before and during all rapid eye movements; tonic units active in relation to deviation of the eyes and to velocity of slow eye movements; and pausing units silent during saccades and firing during smooth pursuits or fixations (73). It is therefore proposed that PPRF is the common integrator of supranuclear inputs to the oculomotor system (105). Thus, the motor output in the eye movement system is generated in the brain stem while the cerebellum exerts control over this output.

CLINICAL STUDIES

Systematic qualitative (20, 28, 29, 59, 61, 99, 116, 134) and quantitative (9, 13, 14, 15, 16, 51, 91, 127, 144, 149, 150) studies on defects in human saccades and smooth pursuits in relation to defined lesions in various parts of the nervous system have been conducted mainly during the last two decades. The findings indicate that disorders in saccades and smooth pursuits may accompany lesions at different sites in the central nervous system (CNS).

In lesions involving supratentorial brain structures, decreased saccadic accuracy and velocity have been reported in patients following hemidecortication (120, 131). However, normal saccadic velocity has been found in patients with frontoparietal lesions (9). Impairment of smooth pursuit directed towards the lesion has been reported after hemidecortication

(120, 132) and after frontoparietal infarction (15), but in both directions after lesions in the occipital cortex (51, 134).

In lesions of the posterior fossa the anatomical proximity and the vascular supply of the cerebellum and the brain stem predisposes to combined cerebellar and brain stem disturbances. In these cases, decreases in the velocity of saccades and smooth pursuits (9) and saccadic dysmetria can occur (35). However, a combination of normal saccades with asymmetric smooth pursuit has also been reported (16, 147), in a rare disorder involving pons and cerebellum (Arnold Chiari malformation). In disorders restricted to the cerebellum impairment of smooth pursuit (41, 134) and dysmetric saccades (35) has been observed. In degenerative diffuse lesions of the posterior fossa such as spinocerebellar degeneration and progressive supranuclear palsy (8, 137, 148), saccades may be abnormally slow and inaccurate. The same findings have been reported in cases of lesions of the medial longitudinal fasciculus (13), i.e. internuclear ophthalmoplegia.

The vestibular disorders may cause transitory bias in smooth pursuit (9) or in optokinetic nystagmus (34, 62, 76, 117) and error of egocentric orientation (63).

Diffuse disorders of the CNS may be accompanied by prolonged saccadic latencies (9), disturbed smooth pursuit (51), fixation instability (42) and disturbances of OKN (40, 45, 92). Similarly, drugs affecting the CNS can impair the smooth pursuit (60, 107) and decrease velocity of saccades (2, 3, 49).

PURPOSE OF THE STUDY

Certain problems in the interpretation of results of clinical studies cited above emerge from the fact that generally accepted standards for the oculomotor tests are still not established. The technique of recording of eye movements, the construction of the tests and the analysis of the results vary from study to study. Consequently, the data obtained in various laboratories may not always be comparable. Available reports on studies concerning the relationship between the fast phases of nystagmus and saccades in man are few and do not allow definite conclusions. Fast phases of nystagmus have not been the object of much interest in clinical studies. Even if the neuroanatomical pathways underlying saccades, fast phases of nystagmus and eye movements during visual tracking are not completely understood, the recent information obtained from studies in animals and in patients indicate that disorders of these eye movements can vary according to the site and extent of the lesion. Thus, further studies on saccades, fast phases of nystagmus and tracking eye movements may help us to assess patients with vertigo.

The study consists of four papers whose purposes were:

- 1) to compare peak velocities of saccades and of fast phases of various types of nystagmus in normal subjects (I);
- 2) to study the velocity patterns of the two types of rapid eye movements as well as saccadic accuracy in patients with various types of lesions (II);
- 3) to quantify the ability to track a visual object travelling at a constant velocity in normal subjects (III);

- 4) to study tracking eye movements in patients with various types of CNS lesions by using the quantitative parameters (IV).

Materials and Methods

SUBJECTS

Studies on saccades and fast phases of nystagmus (I, II)

Normal subjects: 20 volunteers with normal vision and neither previous history of neurological disease nor pathological findings on routine otoneurological examination were included. They were 10 males and 10 females with ages ranging from 23 to 72 years (mean 48).

Patients: 26 patients were selected, affected by any of four different types of neurological disorder: 6 with vestibular neuropathy, 7 with left frontal lobe lesions, 5 with diffuse encephalopathy (meningo-encephalitis) and 8 with lesions within the brain stem. Clinical diagnoses, age distribution and duration of symptoms are given in Table I.

Studies on tracking eye movements (III, IV)

Normal subjects: 20 volunteers with normal vision and neither previous history of neurological disease nor pathological findings on routine otoneurological examination were included. They were 10 males and 10 females with ages ranging from 22 to 70 years (mean 49.9).

Patients: 24 patients were selected affected by any of the four types of neurological disorder: 6 with vestibular neuropathy, 6 with lesions within the left frontal lobe, 6 with lesions in the cerebellum and

Table I. Classification of patients included in studies on saccades and fast phases of nystagmus.

Group	No. of pats.	Diagnosis	No. of pats.	Age (years)		Duration of symptoms
				mean	range	range
Vestibular	6	Vestibular neuropathy	1	47.7	19-72	21 days - 25 years
		Morbus Ménière	3			
		Sudden deafness	2			
Frontal lobe	7	Head injury	2	42.7	26-73	2 months - 7 years
		Infarction	4			
		Presenile dementia	1			
Diffuse encephalopathy (meningo-encephalitis)	5	Mainly pontine	2	38.6	14-58	6-48 days
		Disseminate	3			
Brain stem	8	Infarction	3	48.8	17-75	14 days - 12 years
		Tumour	1			
		Progressive supranuclear palsy	2			
		Amyotrophic lateral sclerosis	1			
		Heredo-degenerative disease	1			

Table II. Classification of patients included in studies on tracking eye movements.

Group	No. of pats.	Diagnosis	No. of pats.	Age (years)		Duration of symptoms
				mean	range	range
Vestibular	6	Vestibular neuropathy	6	57.7	40-69	6-30 days
Frontal lobe	6	Infarction	5	61.8	38-73	2-48 months
		Presenile dementia	1			
Cerebellar	6	Tumour	2	46.0	12-72	3 weeks - 10 years
		Encephalitis	1			
		Degenerative disease	2			
		Infarction	1			
Brain stem	6	Infarction	4	66.0	46-72	1 week - 6 months
		Progressive supranuclear palsy	2			

6 with lesions in the brain stem. Clinical diagnoses, age distribution and duration of symptoms are given in Table II.

METHODS

Recording of eye movements (I-IV)

Eye movements were recorded with direct current electro-oculography (EOG) technique using an ink jet writer (Mingograph M81, Siemens Elema, Stockholm, Sweden) and filter with an upper cut-off frequency of 15 Hz. The paper was fed at a speed of 100 mm s^{-1} during recording of saccades and nystagmus, whereas during the tracking test the paper velocity was 25 mm s^{-1} . Calibrations of eye movements were made using two pairs of light diodes subtending angles of 20° and 60° . The superficial layer of the keratin of the skin was removed with a drill. Commercial silver-silver chloride electrodes and jelly were used (Medicotest^R). The horizontal eye movements were recorded binocularly and the vertical ones monocularly.

Test procedures (I-IV)

The subject was sitting comfortably in a chair with head supported in such a position that the eye-ear axis was horizontal. During the tests verbal contact was maintained with the subject. In addition to EOG, the eye movements could be monitored with an infrared TV camera. The subject was instructed before the test to avoid blinking and to perform the desired eye movements as accurately as possible.

Testing of saccades (I, II)

The visual targets were light diodes of different colours, mounted on a horizontal bar, placed 120 cm

in front of the subject. The pairs of diodes could be switched on-off so that gaze shifts of 5, 10, 20, 30, 40 and 60° could be executed. The subjects were asked to move their gaze as quickly as possible between the two actual diodes. The tests were conducted in light, in darkness, and to imaginary target, with closed eyes.

Testing of fast phases of nystagmus (I, II)

Caloric test with water temperatures of 30°C and 44°C was performed in darkness with the eyes open.

A rotatory test was conducted in a chair by an angular acceleration of 120°s^{-2} for 1 s in the horizontal plane. After 60 s the chair was brought to a standstill by a corresponding deceleration. This test was also carried out in darkness with the eyes open.

Full-field stimulation with an optokinetic drum, consisting of black and white stripes, accelerated for 10 s to a constant velocity of 90°s^{-1} , was used to elicit the optokinetic nystagmus.

Optovestibular nystagmus was induced during rotation as in the rotatory test, within stationary optokinetic drum, in light with eyes open.

Testing of tracking eye movements (III, IV)

The target for eliciting tracking eye movements was a light spot 3 cm in diameter, projected onto a screen placed 160 cm in front of the subject, and moving at various constant velocities of 10, 20, 30, 40, 50 or 60°s^{-1} . The excursion of the target on the screen covered an amplitude of 60° . As soon as the target disappeared in one direction the new one became visible on the opposite edge of the screen. Extreme positions of the target on the screen were detected by photo-

transistors and recorded. At each velocity and direction five trackings were recorded.

ANALYSIS OF RECORDINGS

Saccades and fast phases of nystagmus (I, II)

Saccades and fast phases were classified in six categories, with mean amplitudes of 5, 10, 20, 30, 40 and 60°.

- Peak velocity of the saccades and fast phases of nystagmus was measured by estimating the tangent of the rapid eye movement (I).
- Accuracy of saccades was measured as an amplitude of the initial saccade. Number and amplitudes of possible corrective saccades were also measured (II).

At least 10 consecutive saccades at each amplitude range to right and left were measured.

Tracking eye movements (III, IV)

- Maximum velocity of smooth pursuit was measured on the part of the recording where smooth pursuit showed its highest velocity, lasting at least 250 ms and starting after an initial velocity adaptation period of 500 ms. The maximum velocity gain of smooth pursuit was calculated as the ratio maximum eye velocity/target velocity.
- Total amplitude of tracking was measured as total excursion of the eyes from the beginning of each tracking to the start of the next refixating saccade. Amplitude of smooth pursuit was defined as the sum of amplitudes of all smooth pursuit sequences during the tracking.

- Frequency of superimposed saccades of at least 3° of amplitude was estimated. The superimposed saccades were classified in three categories depending on their amplitude: $3-10^{\circ}$, $11-20^{\circ}$, and 21° and larger. The frequency at each target velocity and direction was extrapolated for a time event of 60 s.
- Square wave was defined as two saccades directed counter to each other and separated by an event of relative standstill. The frequency of square waves was extrapolated for a time event of 60 s at each target velocity and direction.

At each velocity and direction five trackings were analysed.

STATISTICS

The significance of the difference between data displaying different test conditions or different patient groups was tested by using a two-way analysis of variance (ANOVA). The difference between observations was further tested with Student's t-test for independent samples to reveal the degree of difference at individual eye movement amplitude or target velocity. When examining repeatability of test results in one and the same individual a Student's t-test for paired data was used.

Results

STUDIES ON SACCADDES AND FAST PHASES OF NYSTAGMUS

Normal subjects (I)

The peak velocity of the saccades depended on the amplitudes and visual conditions. Thus the peak velocity of the saccade measured in light increased with the saccadic amplitude, from an average of 180°s^{-1} for a saccade of 5° to about 500°s^{-1} for a saccade of 60° . The saccades in darkness were slower than those in light. The saccades executed behind closed eyes exhibited the lowest velocities.

The mean peak velocity of saccades of 20° and 60° in 20 normal subjects tested in two separate laboratories with an interval of one hour were very similar. Nor did the mean peak velocity in a separate group of 12 subjects with similar age distribution differ significantly from the results in 20 subjects in the main study. There was no difference between different age groups as regards the peak velocity of the saccades.

Fast phases of nystagmus displayed lower velocities than saccades of the same amplitudes and under the same visual conditions. Fast phases of caloric, rotatory and optokinetic nystagmus were equally rapid, while the optovestibular nystagmus exhibited somewhat higher velocities (Fig. 1).

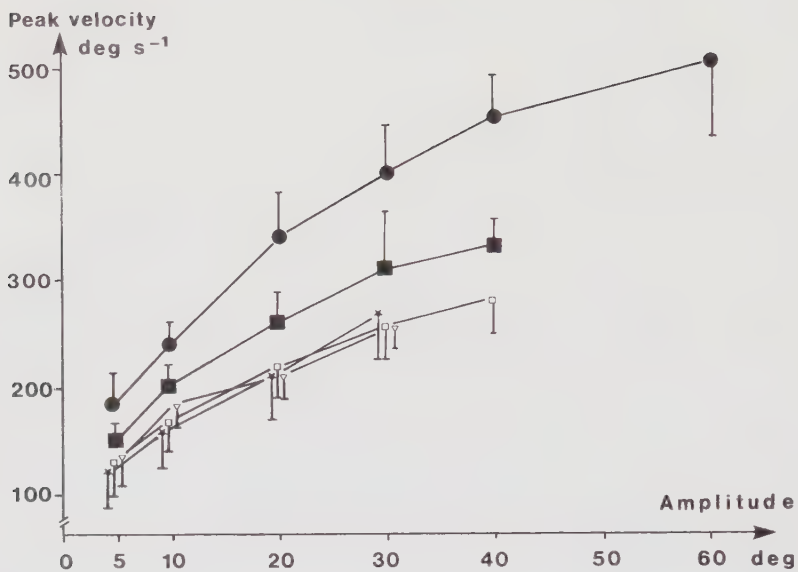


Fig. 1. Peak velocities of visual saccades (●) and fast phases of optovestibular (■), optokinetic (★), rotatory (□) and caloric (▽) nystagmus in normal subjects. Means and standard deviation are shown.

Patients (II)

The peak velocity of the saccades varied according to the site and extent of the pathological condition. In patients with vestibular disorders or lesions within the frontal lobe the peak velocity of the saccades was not affected. In patients with disorders within the brain stem the peak velocity of the saccades decreased significantly, while in patients with meningo-encephalitis there was a significant increase in the peak velocity (Fig. 2a).

The peak velocity of fast phases of rotatory and optokinetic nystagmus varied analogously to the peak velocities of saccades. Thus the values were normal in patients with vestibular and frontal lobe disorders, while in patients with brain stem disorders the values were decreased and in the group with meningo-encephalitis they were increased (Fig. 2b). However, in individual cases, dissociations in the velocity pattern were found between the saccades and fast phases of nystagmus.

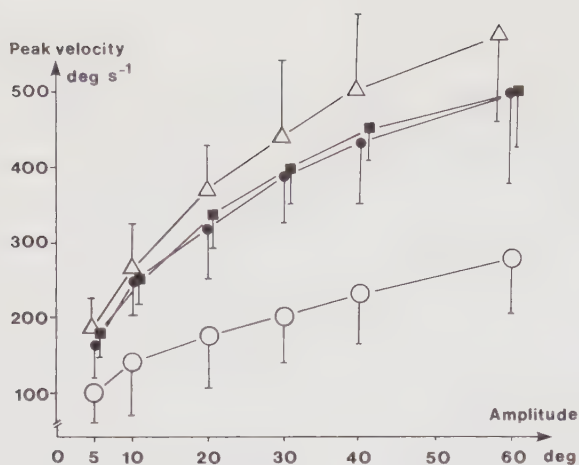
Accuracy of saccades was normal only in the group with vestibular disorders. In the groups with disorders within the central nervous system the initial saccades were usually hypometric (Fig. 2c).

STUDIES ON TRACKING EYE MOVEMENTS

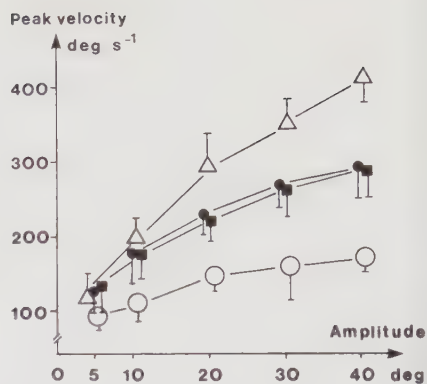
Normal subjects (III)

The maximum velocity of the smooth pursuit corresponded to that of the visual target in only 7.6% of all trackings investigated. The ability to adapt eye velocity to the velocity of the target measured as the maximum velocity gain of smooth pursuit was close to 1.00 at target velocities of $10-40^{\circ}\text{s}^{-1}$ but decreased

A



B



C

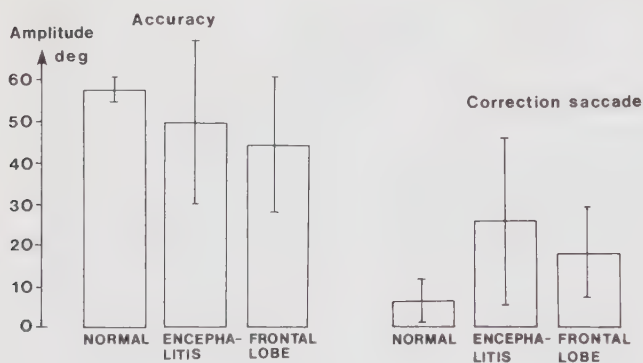


Fig. 2. A) Peak velocities of saccades in normal subjects (■), in patients with disorders of the frontal lobe (●), and brain stem (○), and with meningo-encephalitis (△). B) Peak velocities of fast phases of vestibular nystagmus in the same groups of patients. C) Accuracy and amplitude of correction saccades at target amplitude of 60° in normal subjects, patients with meningo-encephalitis and with frontal lobe disorders. Means and standard deviations are shown.

at higher target velocities (Fig. 3). The interindividual variation was least (15% variation coefficient) at target velocities of $10-40^{\circ}\text{s}^{-1}$ and increased gradually at higher target velocities (to over 30% at a target velocity of 60°s^{-1}).

The total amplitude of tracking and amplitude of smooth pursuit decreased gradually in inverse proportion to the velocity of the visual target (Fig. 3). Smooth pursuit accounted for a major part of the total tracking amplitude at all target velocities.

Superimposed saccades were present in most of the trackings. Only 3 subjects executed tracking entirely free from superimposed saccades. In all trackings analysed the majority of the superimposed saccades (80%) were in the amplitude category $3-10^{\circ}$. Superimposed saccades of amplitudes of $11-20^{\circ}$ (15%) became more prevalent at target velocities of 30°s^{-1} and above, while superimposed saccades of 21° and larger were infrequent (5%), occurring exclusively at target velocities of $30-60^{\circ}\text{s}^{-1}$.

Square waves occurred at low frequencies at all target velocities. In contrast to superimposed saccades, they did not vary significantly in number with the velocity of the target (Fig. 3).

In the group of 9 subjects tested on two occasions the maximum velocity gain of smooth pursuit, the amplitude of smooth pursuit and the frequency of superimposed saccades of $3-10^{\circ}$ were increased at the second test.

Patients (IV)

The maximum velocity gain of smooth pursuit, the total amplitude of tracking and the amplitude of smooth pursuit depended on the site of the lesion (Fig. 4).

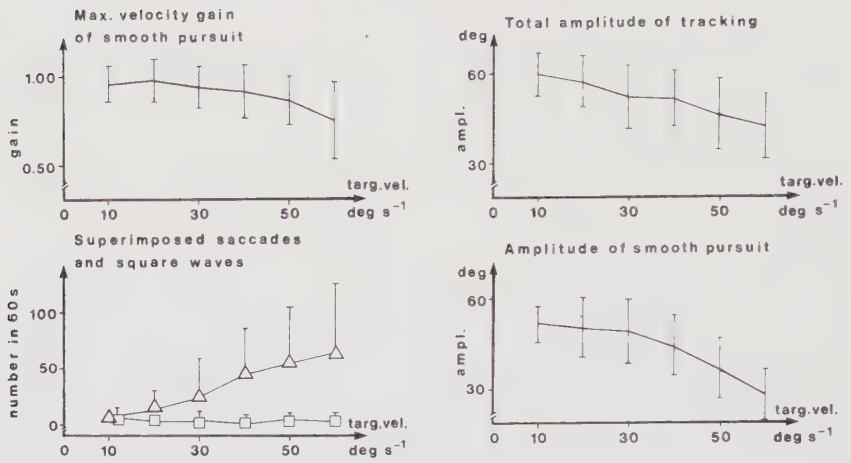


Fig. 3. Maximum velocity gain of smooth pursuit, total amplitude of tracking, frequency of superimposed saccades (Δ) and square waves ($\square$) and amplitude of smooth pursuit at different velocities of the visual target in normal subjects during tracking to the right. Means and standard deviations are shown.

In the group with vestibular disorders the mean values of the maximum velocity gain of smooth pursuit at the respective target velocities were within normal limits. In the group with frontal lobe disorders the values were reduced, especially at target velocities of $40-60^{\circ}\text{s}^{-1}$. Similarly, in the group with cerebellar disorders the maximum velocity gain of smooth pursuit was reduced, especially at target velocities of $30-60^{\circ}\text{s}^{-1}$. In patients with brain stem disorders reduction was found at all target velocities tested.

The total amplitude of tracking as well as amplitude of smooth pursuit were within normal limits in the group with vestibular disorders but reduced in patients with frontal lobe, cerebellar and brain stem disorders.

Superimposed saccades with an amplitude range of $3-10^{\circ}$ occurred more frequently than normal in all groups of patients (Fig. 4).

Only in the cerebellar group did saccades with amplitudes of $11-20^{\circ}$ and 21° and larger appear more frequently than in normal subjects.

The mean frequency of square waves tended to be higher in the four groups of patients compared, however, no significant differences were found between normals and the patient groups.

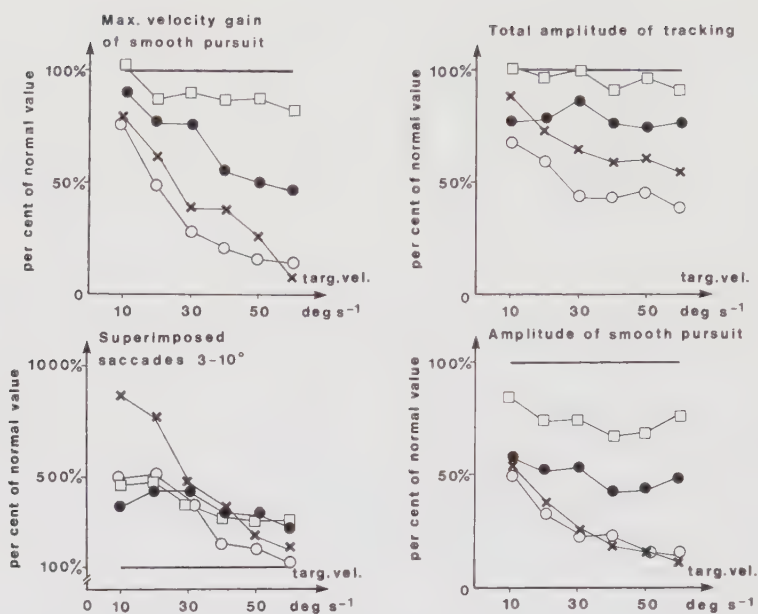


Fig. 4. Maximum velocity gain of smooth pursuit, total amplitude of tracking, frequency of superimposed saccades 3-10° and amplitude of smooth pursuit in patients with vestibular (□), frontal cortical (●), cerebellar (×) and brain stem (○) disorders as percentages of normal (—) values.

Discussion

Analysis of peak velocities of saccades and of fast phases of nystagmus in normal subjects revealed that saccades were faster than fast phases of nystagmus (I). Moreover, there were distinct differences between normals and patients in velocity pattern of rapid eye movements depending on the site of lesion. The peak velocities of saccades and of fast phases of nystagmus could be either decreased or increased vis-à-vis normal subjects (II). Tracking eye movements in patients with disorders of the CNS exhibited deterioration which could be related to the site of lesion (III, IV). The present results support the opinion that analysis of saccades and tracking eye movements with standardized methods is of value for studying the function of the oculomotor system and it should be included in routine otoneurological examination. The following discussion focuses on some principal aspects concerning the methods and the results.

METHODOLOGICAL COMMENTS

Each of the currently used methods for the registration of eye movements is hampered by certain disadvantages (see 145). Electro-oculography was chosen for the present investigation as being suitable for studies on patients. With this technique, distortion of large saccades due to the filter can be expected and a filter with a frequency of 100 Hz was proposed to be the most suitable for recording rapid eye movements (112, 118). In clinical studies, however, filters with lower frequencies are often

chosen (9, 12, 124, 148, 149, 150). In the present study, when testing different filters (I), none was found to distort signals having a rise time of 50 ms or longer. A 15 Hz filter was considered suitable for the present recording purpose in order to avoid high frequency noise. However, a contradictory opinion has been expressed (79, 118).

Analysis of eye movements in patients with defined lesions within the CNS is an accepted method of investigating the human oculo-motor system. It should be emphasized that ongoing healing and compensation within the CNS causes variability in the test results. A lesion at one site may impinge on the surrounding structures or disturb nearby pathways and thus the eye motor disturbances may not always be linked to a single possible lesion site. Account must therefore be taken of possible distant effects of the disorder. In accordance with the aim of the present study pathological cases were selected on the basis of pronounced clinical signs indicating as distinctly as possible the topography of the lesions. However, depending on the etiology, some overlapping between various groups could not be avoided.

Stabilization of the head when testing saccades and smooth pursuits is desirable in order to eliminate compensatory input from the vestibulo-ocular reflex. In animal experiments and in studies with human volunteers, bite bars can be used for this purpose. However, in clinical studies such an arrangement is not suitable. Head supports were found satisfactory in the present study (I-IV). Possible undesired movements of the head were observed with a TV camera, enabling immediate correction of head position and exclusion of artefacts from recordings.

Saccadic and tracking tests in the present study were so constructed that the investigated subjects could

predict the applied stimuli. Previously, predictability of stimulus was regarded as an inconvenience (146). Recently, however, it was pointed out that predictability should not be avoided, being a naturally inherent property of the eye motor system (24, 78). Therefore account should be taken of the increase in velocity gains of smooth pursuit when comparing different studies with a predictable versus non-predictable mode of tracking (126, 146). Similarly, it must be kept in mind that corrective saccades are more frequent and larger in predictable than in non-predictable tests (19, 54), whereas velocity of the saccades is not dependent on predictability (43).

In the saccadic and smooth pursuit tests in the present study, displacement of the eye movement by 60° and tracking the visual target at velocities above 40°s^{-1} were required. Such a displacement and such velocities are beyond the optimal physiological limits for saccades and smooth pursuits. Nevertheless, these should be included in test batteries, since defects of the eye movement system may be better revealed when testing procedures cause the system to operate above physiological thresholds (11, 72, 135).

SACCADES AND FAST PHASES OF NYSTAGMUS

The velocity of saccades was higher than the velocity of fast phases of nystagmus at the same amplitude range (I). To the author's knowledge, there is no previous similar report and the available papers offer rather conflicting information: fast phases of nystagmus were found to be either equally rapid (36, 119) or faster than saccades (115) or else saccades and fast phases of OKN were equally rapid but both were faster than the fast phases of vestibular nystagmus (96). The difference in velocity patterns of rapid eye movements between present study (I, II) and other

studies cited above is difficult to explain. Some variability may be the result of differing recording technique and test conditions. Also alertness (115), fatigue (6) or drugs affecting on the CNS (3) may cause variation in the results.

The reason for velocity difference between saccades and fast phases of nystagmus could be due to neurophysiological mechanisms. Neuronal activity during rapid eye movements consists of two phenomena taking place simultaneously: facilitation of agonistic extra-ocular muscles and inhibition of the antagonistic extra-ocular muscles (27, 103, 121). Identical activity has been recorded in the very same brain stem units during fast phases of nystagmus as during voluntary saccades (73). Thus facilitation steered by paramedian pontine reticular formation (PPRF) seems to be similar (111). During the fast phase of nystagmus, four types of neurons are involved in the activation of motoneurons in nucleus abducens; two types display facilitation and two inhibition (57, 58, 122). During the ipsilateral fast phase the motoneurons of nucleus abducens display facilitation and disinhibition and during the contralateral fast phase disfacilitation and inhibition. Hence, the output of abducens neurons during the fast phase of nystagmus will be, in a mathematical sense, a result of integrated activity of facilitation and inhibition. Saccades, on the other hand, are believed to operate entirely by PPRF (111), making the output on the motoneurons simpler. The neural mechanisms of facilitation and disinhibition of motoneurons during fast phases of nystagmus and saccades might provide a basis for the difference in velocity of the two types of eye movement.

Furthermore, simultaneous with the alerting effect of light, volition itself may be a specific factor facilitating saccade, while in reflexive fast phases of nystagmus such facilitation is apparently lacking,

even in light.

In agreement with several other studies (7, 44, 68, 109, 133, 138, 145) the present study (II) indicates that even in normal subjects the large saccades (above 20°) are often hypometric and a correction saccade is necessary to reach the visual target.

Saccadic dysmetria beyond the normal limits was found in the present study in all groups of patients with a disorder in the CNS examined (II), though in varying degree. Patients with lesions within the frontal lobe exhibited prominent saccadic hypometry and also an increased number of correction saccades. Our findings are consistent with other reports showing pathological hypometry of saccades in disorders at any level of the oculomotor system, viz. the cortex (9, 120) the superior colliculi (56), the basal ganglia (9, 32) or the cerebellum (4). In cerebellar lesions hypermetry of saccades can also occur (35) even though hypometry is more common (10, 149).

As mentioned in the introduction (p.8) the absence of presaccadic activity in the frontal eye field in animal experiments does not necessarily prove that such activity is absent in man. Consequently, when discussing participation of frontal eye field in triggering voluntary saccades the human data should be taken into consideration. The present findings (II) indicate that a unilateral lesion in the frontal eye field produces saccadic dysmetry, while the velocity of the saccades remains unchanged. These findings support the idea that the frontal eye field may participate in the triggering of the voluntary saccades (48). However, a similar saccadic inaccuracy could also occur if the frontal eye field would only monitor the saccades, thus only exerting control over the saccadic system (65). The present results do not give definite support for either of the above mentioned mechanisms.

It is quite obvious, however, that the supranuclear neurons in the brain stem (73) operate quite independently of cortical control with respect to executing of the peak velocity of the saccades.

The decrease in velocity of the rapid eye movements in the group with brain stem lesions was a consistent finding and can be interpreted along the lines of recent electrophysiological findings reviewed in the Introduction (p.11). The brain stem, especially the paramedian pontine reticular formation, is of the utmost importance in triggering a saccade with proper velocity. A lesion at this site of neuronal convergence would be expected to reduce the velocity of saccadic eye movements as well as of the nystagmic fast phases, as, in fact, was observed.

No increase in saccadic peak velocity or nystagmic fast phases has previously been reported in patients, such as were found in the present study (II) in patients with meningo-encephalitis. The meningo-encephalitis group could not be precisely defined with respect to topography of the lesion. Therefore, the finding can be considered only as a sign of functional disturbance of the brain. The increase in saccadic velocity has only recently been confirmed in normal subjects exposed to industrial solvents (81). In these subjects, as in the patients with meningo-encephalitis (II), the lesion is not limited to one particular brain structure - the disturbance is more widespread. Our results do not allow of definite conclusions regarding the mechanism causing increased peak velocity of saccades and of fast phases of nystagmus. However, lack of cerebellar (108) or cortical control, normally exerted on brain stem mechanisms may explain the finding, as also was suggested in the study reporting the effects of industrial solvents (81).

Clinical analysis of rapid eye movements may thus be helpful in differential diagnosis of patients with cortical or brain stem disorders. It may often be sufficient to determine merely the velocity of saccades in order to estimate rapid eye movements. However, dissociations between the different types of eye movement can sometimes occur in one and the same patient. Therefore, additional information may be obtained by analysing the fast phases of nystagmus, too.

TRACKING EYE MOVEMENTS

According to the current concept, the oculomotor system processes information on the target's position and velocity (110, 112). Accuracy of tracking will thus depend on cooperation between the saccadic and the smooth pursuit systems (44). The smooth pursuit system functions optimally within rather low velocity limits (106). In the present study, the physiologic range of smooth pursuit, i.e. when the velocity of the eyes could match the velocity of the target, was between 10 and 40°s^{-1} , confirming the data published in other studies (11, 132, 135, 140).

A decrease in smooth pursuit velocity gain will increase the positional error, which is a stimulus for superimposed saccades (111). In normal subjects in the present study (III) the saccadic component increased markedly at velocities above the physiological limits for smooth pursuit, while in disorders of the CNS, saccades appeared abnormally frequently even at physiological target velocities (IV). In this sense, the increase in the number of superimposed saccades may reflect the degree of deterioration in the maximum velocity gain of smooth pursuit.

It should be pointed out that the smooth pursuit system is not very precise. In agreement with other

reports (95, 141) maximum velocity gains of smooth pursuit of 1.00 occurred in present study (III) in only a minority of the trackings. An explanation for this, beyond the inaccuracy of the neurophysiological mechanisms, may be fluctuation in attention and motivation. In some subjects in the present study (III) keen motivation or learning could be the reason for achieving the maximum velocity gain of smooth pursuit of 1.00 even at target velocities of 50 and 60°s^{-1} . Such findings have also been reported by others (31, 34, 47, 77, 114).

The velocity gain of smooth pursuit may be reduced as a consequence of a disruption of retinal feedback, depression of tonic units within the brain stem, especially in PPRF, a disorder of the cortical control of eye movements, or any combination of these factors. Furthermore, fatigue (34), drugs (107), or organic disorders in the CNS (9, 15, 41, 51, 120, 134) may reduce the velocity gain of the smooth pursuit. The degree of impairment, however, varies according to the site of the lesion, as indicated by the present results (IV). For instance, patients with disorders within the posterior fossa displayed an appreciably reduced maximum velocity gain of smooth pursuit at all target velocities, whereas patients with frontal lobe disorders exhibited a decrease in the values only during the tracking at velocities above physiological limits.

The total amplitude of tracking varied between different groups of patients even though the mean numerical range of the superimposed saccades was similar in different groups of patients (IV). The total amplitude of tracking was normal in the vestibular disorder group in which the increase in the saccadic component compensated for the loss of smooth pursuit during the tracking. In patients with frontal lobe disorders, an increase in saccadic compensation

resulted in a mere 25% decrease in normal total amplitude of tracking. In patients with brain stem and cerebellar lesions the smooth pursuit component was severely disturbed and despite saccadic compensation the total amplitude of tracking was decreased by 40% or more.

The analysis of tracking eye movements in different groups of patients illustrates the functional organization of the oculomotor system. The system appears most susceptible to disturbances in the brain stem and cerebellum where the eye motor impulses are integrated with the multisensory inputs (53, 105). The principal differences between the cerebellum and the brain stem structures indicate that lesions within the brain stem may cause disturbances in the performance of the eye motor function, whereas the cerebellar disorder may disturb the precision of the eye motor function. In the cerebellar disorders deterioration of smooth pursuits could be compensated to some degree by an increase in the saccadic component of tracking, whereas in the patients with brain stem disorders this compensation was limited.

Frontal lobe disorders caused fewer pathological disturbances of the tracking, thus supporting the opinion that this part of the brain is probably less involved in controlling this type of eye movement (21, 65). In vestibular disorders, only a slight decrease in smooth pursuit appears to be well compensated by the saccadic system.

Conclusions

1. Peak velocities of saccades were faster than peak velocities of various types of nystagmus at the same amplitudes, indicating that in man the two types of rapid eye movement are partly mediated by separate mechanisms.

2. The peak velocities of saccades and fast phases of nystagmus were decreased in patients with brain stem disorders and increased in meningo-encephalitis. In patients with frontal lobe disorders normal peak velocities of the saccades were combined with a decrease in accuracy of the voluntary saccades. The findings indicate that the velocity of the rapid eye movements is essentially linked to the brain stem structures and dependent on modulation by cortical and cerebellar influences.

3. Voluntary tracking was quantified using various parameters. In normal subjects during tracking at velocities between 10 and 60°s^{-1} the smooth pursuit system is predominant, though supported by the saccadic system.

4. The parameters of tracking were slightly affected in vestibular disorders, moderately in frontal cortical disorders and severely in cerebellar and brain stem disorders. The validity of the tests, in particular those designed to reveal disorders of the brain stem and cerebellum, seems to be satisfactory.

The saccadic and tracking tests should be incorporated into the routine otoneurological examination in order

to support the differential diagnosis between peripheral and central disorders causing vertigo.

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VELOCITY PATTERNS OF RAPID EYE MOVEMENTS

N. G. Henriksson, I. Pyykkö, L. Schalén and C. Wennmo

From the Department of Otorhinolaryngology, University Hospital, Lund, Sweden

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Abstract. The peak velocities of saccades and fast phases of nystagmus were examined and compared in 20 healthy subjects. The peak velocities of both types of eye movements increased with increase of amplitudes. The saccades were found to be fastest in light, slower in darkness and slowest behind closed eyelids. The peak velocities of the quick phases of optokinetic and of vestibular nystagmus were found to be the same. Fast phases of optovestibular (optic as well as vestibular stimulation) nystagmus produced significantly higher peak velocities than the two others. At the same amplitude and during the same visual conditions the saccades were significantly faster than any type of fast components of nystagmus. The difference in velocity between voluntary and reflexive eye movements is possibly related to differences in antagonistic activity during these eye movements, but also to specific synaptic events during the voluntary action.

There are two main groups of rapid eye movements, the saccades and the fast phases of nystagmus. Whereas the saccades can be triggered voluntarily or spontaneously by peripheral visual stimuli, the fast phases of vestibular and of optokinetic nystagmus are triggered reflexively, secondary to the slow phase of nystagmus.

All rapid eye movements seem to utilize the same pontine neuronal network located in the parapontine reticular formation (Cohen & Feldman, 1968; Cohen & Komatsuzaki, 1972; Cohen et al., 1973; Henn & Cohen, 1975), where the position of the eyes is adjusted to the position of the target (Robinson, 1975). A similar firing pattern is thus found in these neurons during saccades as well as during fast phases of nystagmus (Keller, 1974; Henn & Cohen, 1975).

This would support findings by several authors (Dichgans et al., 1969; Nauck et al.,

1969; Ron et al., 1972) who neither in man nor in intact alert monkey found differences between saccadic velocity and velocity of fast phases of nystagmus.

As these findings were in contrast to our observations the present study of the velocity of the two types of rapid eye movements was undertaken.

The aim of this paper will be to determine the velocities of the fast phases of nystagmus of 20 normal subjects exposed to rotation, to caloric stimulation as well as to optokinetic stimuli, for comparison with the velocity of the voluntary saccades at corresponding amplitudes.

MATERIAL AND METHODS

The material consisted of 20 volunteers with normal hearing and vision. They had no history of neurological disease and a routine otoneurological examination revealed free eye movements. Ten were males and 10 females and their mean age was 48 years with a range of 23 to 72 years. An electro-oculographic (EOG) technique with commercial electrodes and electrode jelly (Medicotest®) was used.

In the horizontal plane the mean of the movements of the right and left eye was recorded by the use of conventional electrodes, horizontally disposed, while the recording of vertical eye movements was made for each eye separately. A DC-coupled ink-writer with an upper cut-off frequency of

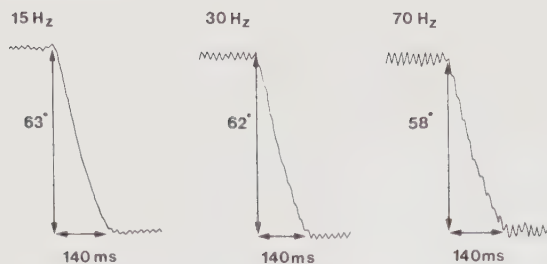


Fig. 1. Changing the lowpass filters from 15 to 30 and to 70 Hz does not significantly influence the velocity of the saccade as calculated from the recordings.

15 Hz (Mingograph M 81, Siemens-Elema, Stockholm, Sweden) served as a recorder.

Before the selection of the filter was made, the rise time of the amplifier was tested by using square waves and ramp signals corresponding to different angular velocities of the eyes. When filters of 15, 30, 70 or 700 Hz were used, the amplitude, the shape, and the duration of these "electrical saccades" did not change noticeably when signals with a rise time of 50 ms or longer were applied. To exclude high frequency noise we could therefore use the 15 Hz filter without any distortions of the signal (see also Fig. 1).

The paper was fed at $100 \text{ mm} \cdot \text{s}^{-1}$ to

secure the accuracy in the analysis of recordings for events occurring within 10 ms (1 mm). In the recording an angular deviation of the eyes of 1° corresponded to 1 mm.

The patient was sitting comfortably in a chair. The head was mechanically supported and the position of the head adjusted (eye-ear axis horizontal) for each subject before any measurements was undertaken. Light diodes were placed 120 cm in front of the subject at angles of 5° , 10° , 20° and 30° to the right as well as to the left of the mid-line. Calibration was made with the subject looking alternately at the diodes which were 20° and 60° apart.

Saccades. The tests were performed in darkness. The targets consisting of light diodes of different colours were therefore easy to distinguish. At least 10 saccades at each of the amplitudes 5° , 10° , 20° , 30° , 40° and 60° were performed. The light diodes were then switched off and the subject was asked to look first with his eyes open and then to try to look also with his eyes closed at the diodes 10° apart, 20° apart, and finally, at those 60° apart.

Caloric test. A routine caloric test (Hen-

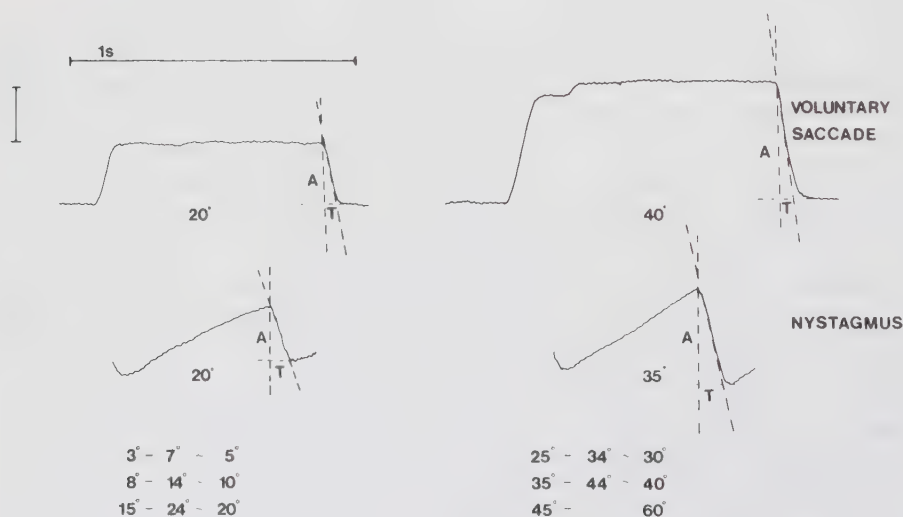


Fig. 2. The peak velocities of rapid eye movements are measured and classified according to amplitude of the eye movements. The tangent at peak velocity is visually

determined for the recording and is calculated ($pV \times A/T$) by using the amplitude (A) and duration (T) at peak velocity (pV).

Table I. Mean peak velocities with spreads of voluntary saccades at amplitudes of 20° and of 60° in two groups of subjects tested in two laboratories

	No.	Age (years) mean and range	Laboratory	Peak velocity of saccades at	
				20° mean $\pm$ S.D.	60° mean $\pm$ S.D.
Group A	20	48.4 (23–72)	I	336° s ⁻¹ $\pm$ 42° s ⁻¹	495° s ⁻¹ $\pm$ 85° s ⁻¹
			II	327° s ⁻¹ $\pm$ 61° s ⁻¹	480° s ⁻¹ $\pm$ 96° s ⁻¹
Group B	12	45.0 (18–70)	II	316° s ⁻¹ $\pm$ 67° s ⁻¹	465° s ⁻¹ $\pm$ 96° s ⁻¹

riksson et al., 1972) was conducted in darkness with the subject's eyes open.

Rotation test. The rotation test was performed in complete darkness by acceleration of the subject within one second to a constant velocity of 120° s⁻¹. After one minute of rotation the chair was brought to a standstill within one second. The perrotatory as well as the postrotatory nystagmus was recorded with the eyes open.

Optokinetic test. The optokinetic test was performed with the subject inside a striped drum rotating around the patient. The drum was accelerated for 10 seconds to a constant velocity of 90° s⁻¹ and the calculations were made at velocities of the drum at 90° s⁻¹ as well as somewhat below this value.

Optovestibular test (optokinetic + rotation stimulus). In this test the subject was rotated inside the optokinetic drum with his eyes open. Using this technique eye movements were recorded resulting from a combination of vestibular and optokinetic stimuli. The physical properties of the acceleration in this combined test were the same as those in the rotation test in darkness.

Evaluation of recordings. The peak velocities were measured by estimating the tangent for rapid eye movements; this usually occurred at the beginning of the eye movement. The saccades and the fast phases of nystagmus were classified into different groups according to their amplitudes (Fig. 2). The means and standard deviations of the velocities of the eyes in the saccades and in

the fast phases of the various types of nystagmus were determined for each individual and for each amplitude (10°, 20°, 30°, 40° and 60°).

To assess the stability of the mean saccadic velocity within the same group of normals, this velocity was determined on two different occasions with an interval of about one hour. Two different sets of recording devices in two different laboratories were used.

To study the variation between two different groups of normals, another group of 12 subjects was examined with a similar age distribution as the subjects in the main study (Table I).

RESULTS

Saccades. The mean peak velocities of the saccades at amplitudes of 20° and 60° for the same group of subjects in two different laboratories are provided in Table I. Despite the fact that the recordings were made in different laboratories with an interval of one h, the mean peak velocities were very close. Also when two separate groups of subjects were examined the mean peak velocities were quite similar (Table I).

The amplitude—peak velocity relationship of saccades light, in darkness and with closed eyes is shown in Fig. 3. Saccade peak velocity was dependent not only upon the amplitude but also on the visual condition; with a given visual condition, an increase in amplitude produced an increase in the peak velocity, the function being curvilinear.

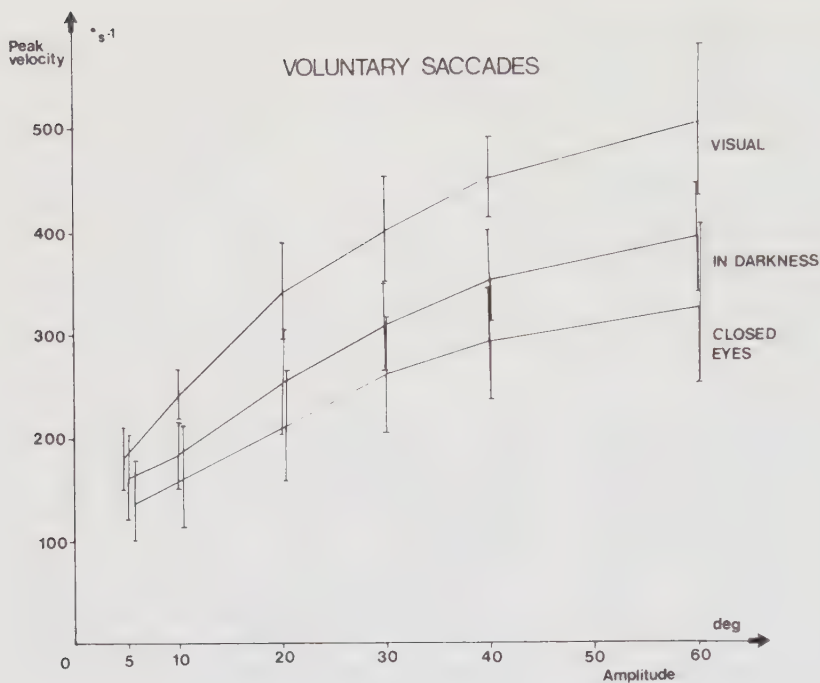


Fig. 3. Mean peak velocities with standard deviations of voluntary saccades when performed in visual conditions, in darkness and in darkness behind closed eyelids at different amplitudes.

At 20° amplitudes the mean peak velocity of the saccades in light was about 340° s⁻¹, in darkness about 250° s⁻¹ and behind closed eyes about 210° s⁻¹ (Fig. 3). From these values the peak velocities increased with increasing amplitudes of the saccades and at amplitudes of 60° were 500°, 400° and 320° s⁻¹, respectively (Fig. 3).

Saccades were characterized by highly variable velocities when performed without visual control. Sometimes efforts to produce saccadic eye movements with closed eyes resulted in recordings appearing more as slow deviations of the eyes than as fast saccadic eye movements (Fig. 4). In darkness, overshooting saccades were very common, especially at the amplitudes of 10° and 20°.

There was further a considerable inter-individual variation in saccadic velocity. Since this study was focussed on the mean values of a whole group of subjects examined in standardized visual conditions, no further interest was paid to either inter- or intraindividual variations.

We found no difference in the peak ve-

locities of saccades between different age groups (Fig. 5). Young subjects had no higher saccadic velocity than older ones; in fact a subject aged 72 had faster saccades than another subject aged 22.

The rapid return eye movements following the pursuit eye movements were analyzed in five subjects. These rapid eye movements had the same amplitude-velocity relationship as visual saccades. Hence, we considered these movements identical to the saccadic ones.

Fast phases of nystagmus. We found no difference in the peak velocities of the fast phases of caloric nystagmus, whether induced by irrigation of the right or of the left ear, by cold or by warm water. These peak velocities were, as were the velocities of the saccades, strongly dependent upon the amplitude of nystagmus as shown in Fig. 6. Moreover, the fast phases of nystagmus were equal, whether caused by rotation or by caloric stimulation. The peak velocities of the fast phases of nystagmus induced by rotation at amplitudes of 20° were about

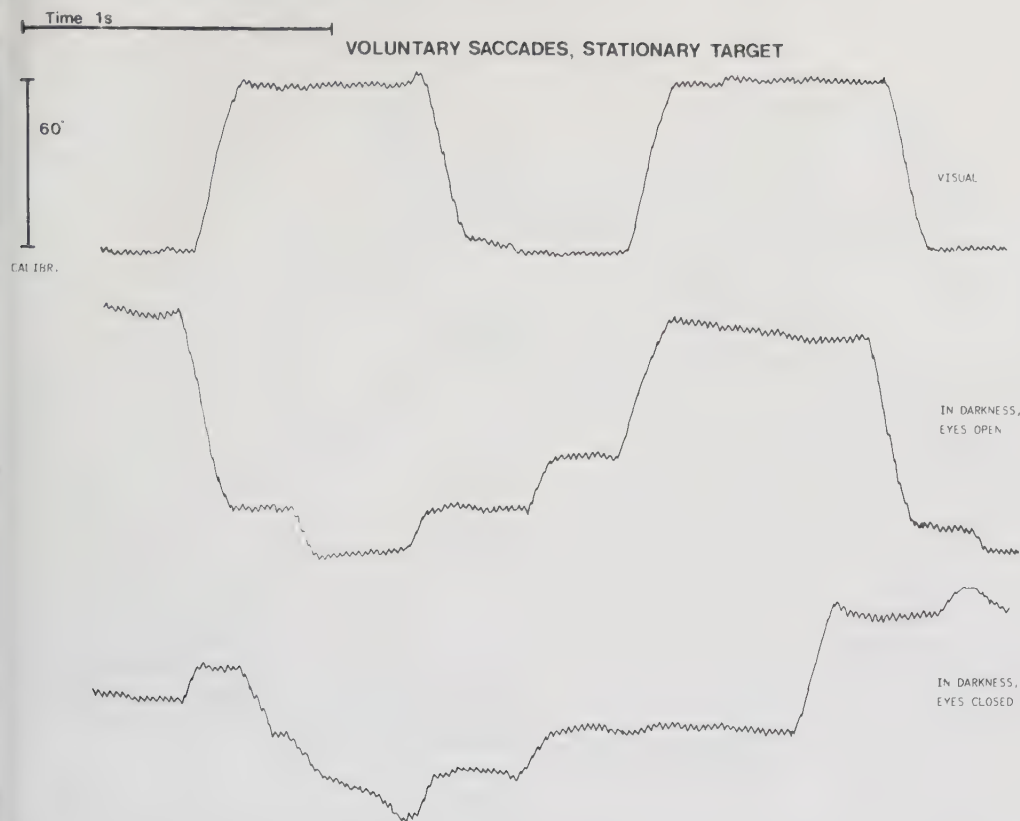


Fig. 4. A typical recording showing voluntary saccades when performed in visual conditions, in darkness eyes open and in darkness behind closed eyelids.

$215^{\circ} \text{ s}^{-1}$. Velocities at higher amplitudes could not be determined, as such nystagmus is rare in adults (Tibbling, 1969).

The amplitudes of OKN (optokinetic nystagmus) were generally smaller, usually below 20° . By increasing the velocity of the stimulus the amplitude of nystagmus tended to decrease. Hence, for optokinetic nystagmus we obtained only a few measuring points for higher amplitudes than 20° . The amplitude-velocity relationship for the fast phases of OKN is also presented in Fig. 6. For the measurable amplitudes the OKN fast phase velocities closely followed the corresponding curves for the fast phase of vestibular nystagmus.

By rotating the subjects within the optokinetic drum with the subject's eyes open

and in light (combining visual and vestibular stimuli) the peak velocity of the fast phase of nystagmus increased (Fig. 6). The peak velocity of the fast phases of the combined nystagmus about $260^{\circ} \text{ s}^{-1}$ and the amplitude of 20° against $215^{\circ} \text{ s}^{-1}$ at the same amplitude for pure vestibular or pure optokinetic nystagmus.

Comparison of velocities of saccades with fast phases of nystagmus. Fig. 7 shows a summarising diagram of the peak velocities of saccades and of fast phases of nystagmus with eyes open in light or in darkness. With the same visual condition and at the same amplitude the saccades were always faster than the fast phases of any type of nystagmus here examined. The difference was more pronounced at greater amplitudes.

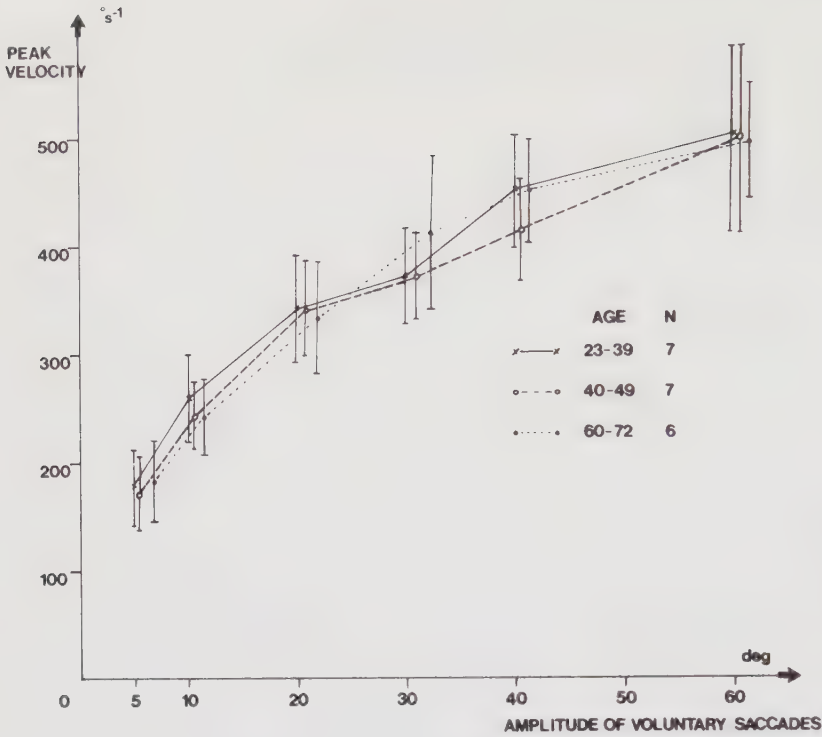


Fig. 5. Mean peak velocities with standard deviations of visual voluntary saccades in three different age groups at different amplitudes.

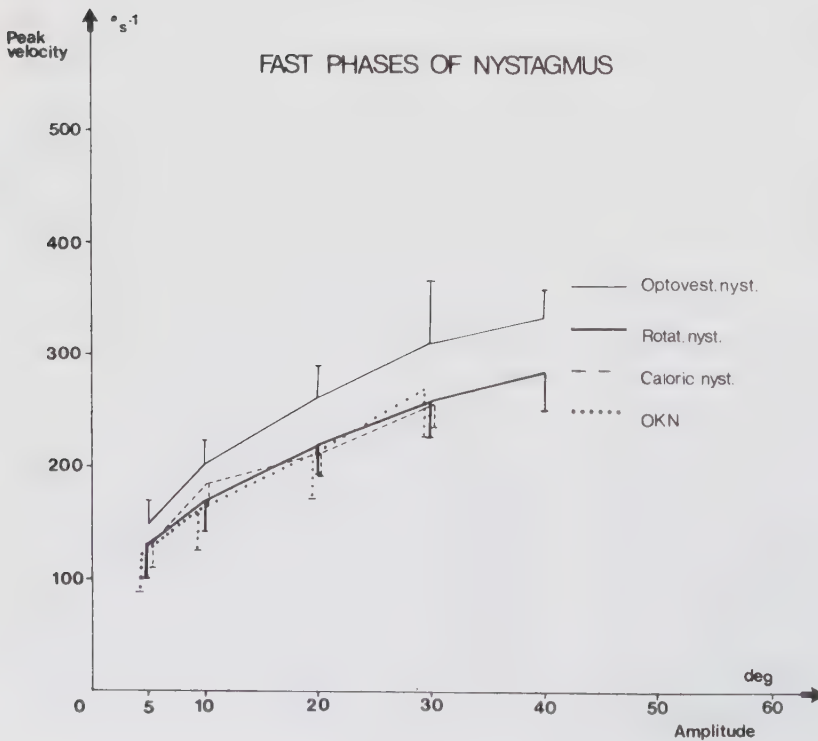


Fig. 6. Mean peak velocities and standard deviations of fast phases of optovestibular nystagmus (—), of rotatory nystagmus (---), of caloric nystagmus (---) and of optokinetic nystagmus (.....). The spreads are presented only in direction.

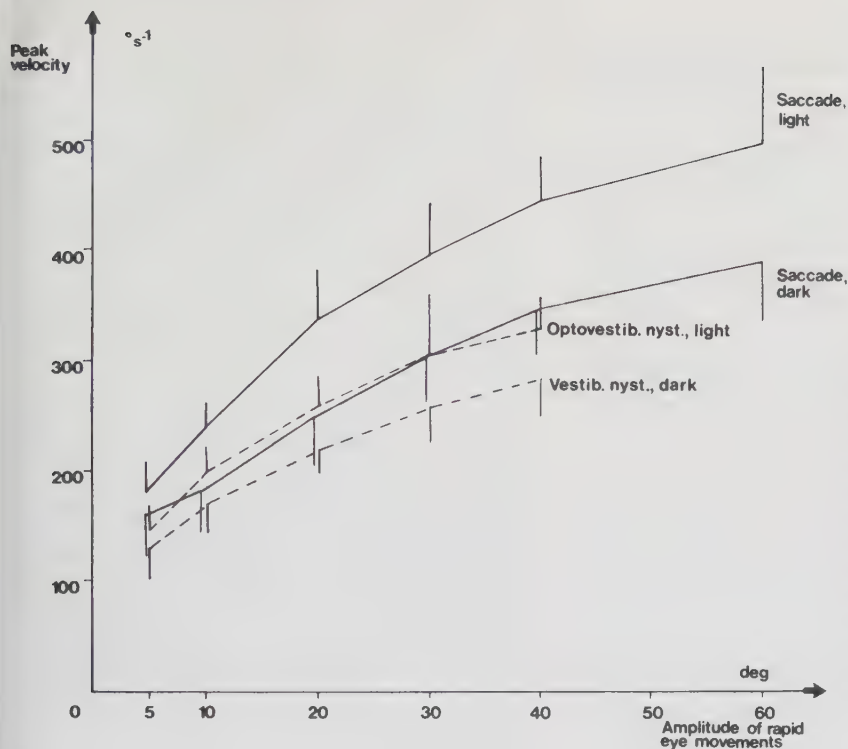


Fig. 7. A summarizing diagram showing the difference between velocities of saccades in light and of fast phases of optovestibular nystagmus. The difference is also shown between the velocities of the saccades in dark and the velocities of fast phases of rotatory nystagmus (vest. nyst. dark). The parameters are as in Figs. 3 and 4.

DISCUSSION

It must be stressed that during the saccade the velocity is not constant. The peak velocity is usually reached somewhat before the middle of the saccades, after which the velocity gradually declined (Westheimer, 1954; Collins, 1975). Hence the saccades represent typical ballistic movements of the eyes (Robinson, 1964). Our reason for measuring peak velocity instead of duration or mean velocity of saccades was due to an observation by Keller (1974) who found a highly synchronous firing pattern within the pontine neurons during peak velocities. We have therefore assumed that peak velocity should be more sensitive to disturbances in clinical connection than other parameters of the saccades (Baloh et al., 1975).

The difference in velocity between saccades and fast phases of nystagmus has not been reported earlier. The complexity in oculomotor mechanism makes the observation difficult to explain. Thus, the cortical

mechanism involved in visual saccades is not yet fully understood. Single unit recordings have shown activity during saccades in the neurons of Brodmann's area 8 (Bizzi, 1968; Bizzi & Schiller, 1970), area 7 (Straschill & Schick, 1974; Mountcastle et al., 1975; Lynch et al., 1977) but also in areas 17, 18, 19 (Hubel & Wiesel, 1965; Orban & Callens, 1977a, b). The importance of these centres is further supported by lesion studies (Pasik & Pasik, 1964; Latt & Cowey, 1971, 1972).

The efferent pathways from these three cortical fields descend to neurons in the pontine reticular formation (Astruc, 1971). Through other pathways the same pontine neuronal network is also activated during the fast phases of vestibular and optokinetic nystagmus. From there oculomotor impulses are channeled to the oculomotor nuclei through final pathways common to all different types of rapid eye movements (Henn & Cohen, 1975). This conception of a final pathway common to the oculomotor system has sup-

ported the idea that all types of rapid eye movements should be equally rapid. Our observations to the contrary may have some support in previous literature also.

Thus, the normal contraction of the antagonistic muscles present only during the fast phase of nystagmus (Shimazu, 1972) might explain a difference in velocity between fast phases and saccades. Further, the "slow" saccadic velocities noticed, e.g. in Huntington's chorea, has been believed to be due to a lack of proper inhibition of antagonistic muscles (Starr, 1967).

One other factor which seems to be of importance is that saccades are initiated voluntarily, but fast phases of nystagmus are released automatically and secondarily to slow phases. During voluntary saccades the subject is alert and active performing a task, while during fast phases of nystagmus the subject is passive. The higher velocity of the saccades might therefore at least in part be explained by a more powerful excitation of pontine neurons caused by volition or related to alertness caused by this volition.

ZUSAMMENFASSUNG

Willkürliche schnelle Augenbewegungen (Saccaden) und reflexbedingte schnelle Augenbewegungen (schnelle Nystagmusphasen) wurden bei 20 gesunden Personen untersucht und miteinander verglichen. Die Geschwindigkeiten beider Augenbewegungstypen stiegen mit einer Zunahme der Amplituden an. Es wurde gefunden, daß die willkürlichen Augenbewegungen, die Saccaden, im Licht am schnellsten, langsamer in Dunkelheit und am langsamsten hinter geschlossenen Augenlidern sind. Während die Geschwindigkeiten der schnellen Phasen von optokinetischen und vestibulären Nystagmen identisch waren, erreichten die schnellen Phasen von opto-vestibulären Nystagmen (sowohl optische als auch vestibuläre Stimulation) signifikant höhere Geschwindigkeiten als beide anderen Typen. Bei gleicher Amplitude und unter gleichen visuellen Bedingungen waren die Geschwindigkeiten der willkürlichen schnellen Augenbewegungen (die Saccaden) signifikant höher als die schnellen Komponenten reflexbedingter Augenbewegungen.

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N. G. Henriksson, M.D.
E.N.T. Department
Hospital of Lund
S-221 85 Lund
Sweden

Rapid eye movements reflecting neurological disorders

N. G. HENRIKSSON, B. HINDFELT, I. PYYKKÖ, AND
L. SCHALÉN.

Division of Otoneurology, Departments of Otolaryngology and Neurology, University Hospital, Lund, Sweden

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Rapid eye movements reflecting neurological disorders

The accuracy and velocity of voluntary saccades together with the velocity of fast phases of nystagmus were studied in 26 patients belonging to 4 groups with defined neurological disorders. Patients with speech disorders of the dyspraxia type, indicating the lesion in the left frontal cortex, showed a pronounced decrease in saccadic accuracy in combination with normal saccadic and quick phase velocity. Patients with pontine disorders showed a significant decrease in the peak velocity of saccades and quick phases of nystagmus while the accuracy of the saccades ranged from normal to severely disturbed. Patients with acute meningo-encephalitis showed increases in peak velocity of saccades and of quick phases of nystagmus and the accuracy was somewhat reduced. Patients with a labyrinthine disease showed normal velocity of saccades and of quick phases as well as normal accuracy of saccades.

Keywords *saccades fast phase of nystagmus CNS diseases labyrinthine diseases*

There are two kinds of rapid eye movements: saccades and fast phases of nystagmus. The former represent eye movements produced either voluntarily or spontaneously. The latter eye movements are triggered reflexly, secondary to slow phases of nystagmus. The neuronal activity related to saccades can be recorded in various cortical areas¹⁻⁵; that related to the fast phases of nystagmus can be studied in the pontine reticular formation.^{6,7}

Following experiments made on alert, intact monkeys, the neurological mechanism of rapid eye movements can be tentatively represented as in Figure 1. During voluntary saccades, neuronal activity takes place in the frontal visual cortex (area 8)^{2,8,9} and in the posterior parietal cortex (area 7).^{3,10} The spontaneous saccades are probably elicited from the primary visual cortex (areas 17, 18, 19).^{4,5} Other researchers¹¹⁻¹⁴ favour the superior colliculi as a possible site of

initiation of saccades. From these higher centres impulses are projected to the pons where the velocity-amplitude adaptation of saccades takes place.⁷

Optokinetic impulses from the occipital visual cortex and vestibular impulses from the labyrinth are projected to the common pontine neuronal network releasing optokinetic and vestibular nystagmus respectively.^{4,5,11,15-17}

The fast phases of nystagmus use, at least partly, the same neurons in the pons as do saccadic eye movements.⁷ This information concerning different triggering of rapid eye movements has not been properly applied in the study of patients with neurological disturbances. In fact in only a few reports have rapid eye movements been subjected to systematic analysis.¹⁸⁻²²

In view of these considerations there should be typical changes in rapid eye movements

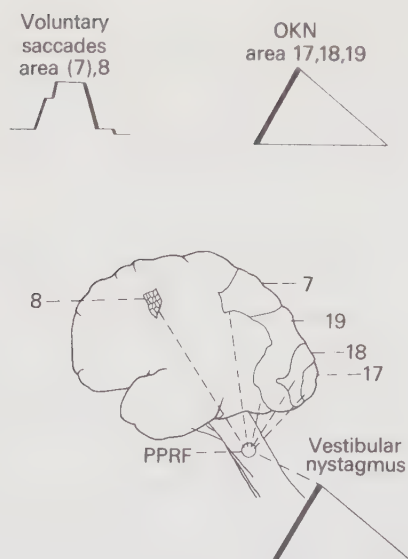


Figure 1 A hypothetical mechanism for triggering of horizontal rapid eye movements (Brodmann's areas of the human cerebral cortex are slightly modified from Ranson & Clark).⁴³

which might be diagnostic of different neurological disorders. In order to test this assumption we have studied 4 groups of patients with various neurological disorders, three of which exhibit a characteristic disturbance of rapid eye movements.

Material and methods

Twenty normal subjects ranging in age from 22 to 64 years (mean 48 years) were recruited for

the determination of normal values.²³ They were healthy with normal neuro-otological findings and free of any illness that might interfere with their performance during the test.

Twenty-six patients with different neurological diseases were selected for this study.

Group 1 Seven patients with dyspraxia of speech,²⁴ indicating disturbance of the motor speech area located in the near vicinity of the left cortical area 8. Three of these patients had infarctions, three had been injured in traffic accidents and one had a degenerative disease of the Niemann-Pick type. Four of these patients have been reported earlier.²⁵

Group 2 Eight patients with a pontine disorder, three of whom suffered from infarctions confirmed by angiography. Three had a degenerative disease of the pons, 2 a progressive supranuclear palsy and one olivo-pontocerebellar degeneration. One had a tumour in the IVth ventricle revealed by computerized axial tomography.

Group 3 Five patients suffered from acute viral meningo-encephalitis. They were diagnosed by typical cell findings in the cerebrospinal fluid. Clinically, various parts of the brain were affected, causing different neurological symptoms and signs. One patient had ataxia with visual dyspraxia, one had signs of pontine

Table 1 Classification of the patients according to disorders and duration of symptoms

Group	Age (yr)			Symptom/diagnosis	n	Duration of symptoms (range)
	n	mean	range			
Dyspraxia of speech (Area 8 disorders)	7	42.7	26-73	head injury	2	2-9 mo.
				vascular ischemia	4	2 mo.-7 yr
				presenile dementia	1	5 yr.
Pontine disorders	8	48.8	17-75	vascular ischemia	3	14-21 d
				tumour in IVth ventricle	1	2 yr
				progress. supranucl. palsy	2	1-12 yr
				aniotrophic lateral sclerosis	1	6 mo.
				heredo-degenerative disease	1	
Encephalitis	5	38.6	14-58	pontine	2	6-48 d
				general	3	13-14 d
Vestibular disease	6	47.7	19-72	vestibular neuronitis	1	23 d
				Ménière's disease	3	6 mo.-25 yr
				sudden deafness	2	21 d-5 mo.

neurone involvement, while three had headache and nausea without focal signs.

Groups 4 Six patients with vestibular disorders. Three had Ménière's disorder, 2 experienced acute hearing loss with peripheral labyrinthine involvement and one had 'vestibular neuronitis'.

The mean age with range, duration of symptoms and diseases of all these patients are provided in Table 1.

DC-electro-oculograms of horizontal and vertical eye movements were recorded simultaneously on a polygraph (M 81, Siemens-Elema, Stockholm, Sweden). A 15 Hz filter was used and the paper was fed at 100 mm s^{-1} . The accuracy of the interpretation of recordings was 10 ms (1 mm) with an amplitude of one degree (1 mm). Standard electrodes (Medioco-test) with jelly were used. The superficial keratin layer was cleansed and removed with a drill. Calibration at angles of 20° and 60° was performed twice, before and in the middle of the test. The subject sat with his head supported and performed voluntary saccades between light diodes placed at angles of 5° , 10° , 20° , 30° , 40° and 60° in front of him. The subjects were asked to change their gaze as fast as they could. No movements of the head were permitted.

Following this, the rotatory test was performed. The rotatory chair was accelerated at 120° s^{-2} for 1 second, rotated for 1 minute and stopped during 1 second and the nystagmus recorded. The optokinetic test was conducted by rotating an optokinetic drum around the subjects with a maximum velocity of 90° s^{-1} .

EVALUATION OF RECORDINGS

The peak velocities of the different types of eye movements at the different amplitudes were measured by determining the maximum inclination which was usually found at the beginning of the movement.

The eye movements were classified into six different groups with the mean amplitudes of 5, 10, 20, 30, 40 and 60° . Measures of the voluntary saccades were taken from the first 10 saccades of each amplitude.

The peak velocities of the fast phases of vestibular nystagmus were calculated during the first 20 seconds of the angular acceleration response and the fast phases of OKN were determined at velocities of the stimulus between 60° s^{-1} and 90° s^{-1} .

In this way a total of about 6500 saccades and fast phases were measured and analysed.

The accuracy of the saccades was determined only for the 60° amplitude between the targets. As in these tests the initial saccade frequently was found to make undershoots and sometimes also overshoots, and as these errors were corrected by one or more 'correcting saccade' it was possible to quantify the accuracy by use of the mean and spread of the amplitude of the initial as well as of the correcting saccade. A value of the initial saccade close to 60° combined with a small spread would then indicate a high degree of accuracy while a high mean of amplitude of the correcting saccade in combination with a large spread would express a low degree of accuracy.

To sum up, the evaluation of the recordings was made with regard to peak velocity of the voluntary saccades, to peak velocity of fast phases of optokinetic and of vestibular nystagmus, as well as to the accuracy of the voluntary saccade (targets 60° apart), expressed as the mean amplitude of these efforts supplemented with the amplitude of the correcting saccade(s).

Results

PATIENTS WITH DYSPRAXIA OF SPEECH

The saccadic velocity of these patients was normal (Figure 2 & 3). The fast phases of nystagmus showed the same peak velocity as those of normal subjects (Figure 4).

The shape of these saccades differed from that of normals. The saccades were split into smaller saccades and usually more than two saccades were needed before the target was reached. Thus, the mean accuracy of the saccades was reduced. The initial saccade only reached an average of 43° of the required 60° between the targets. The need for correction saccades thereby increased (Figure 5).

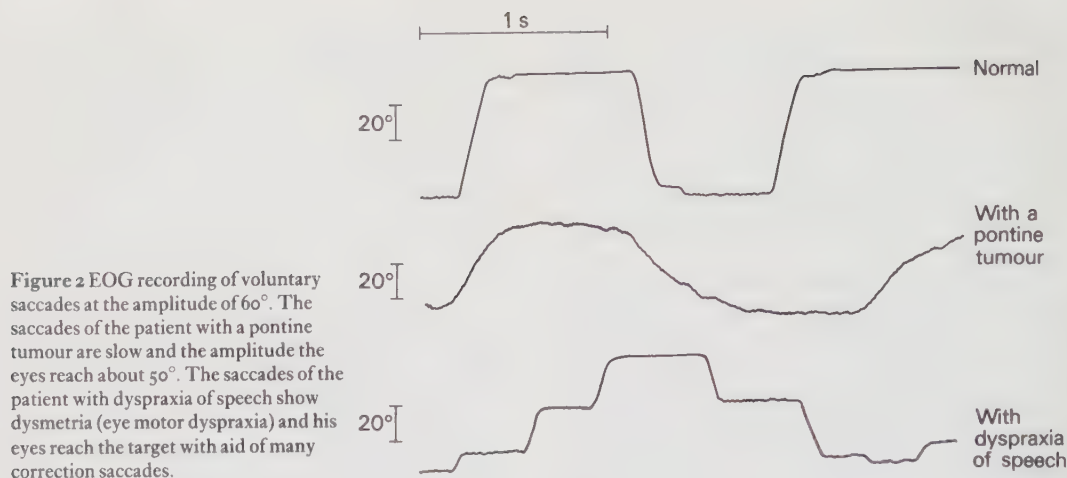


Figure 2 EOG recording of voluntary saccades at the amplitude of 60° . The saccades of the patient with a pontine tumour are slow and the amplitude the eyes reach about 50° . The saccades of the patient with dyspraxia of speech show dysmetria (eye motor dyspraxia) and his eyes reach the target with aid of many correction saccades.

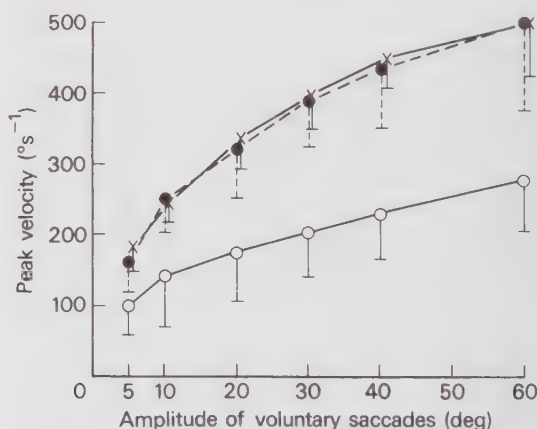


Figure 3 Peak velocities of voluntary saccades in normal subjects (x), in patients with dyspraxia of speech (●) and in patients with pontine disorder (○). Mean and standard deviation are shown in one direction.

PATIENTS WITH PONTINE DISORDER

The mean peak velocity of the saccades was significantly lower in patients with pontine disease than in normals (Figures 2 & 3). In two

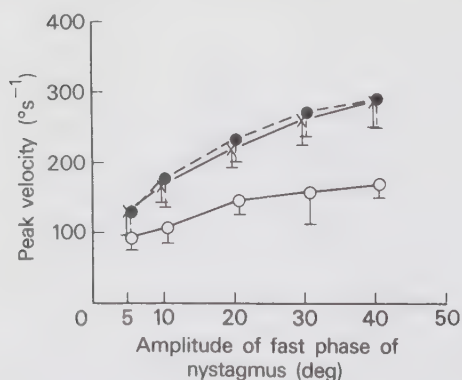


Figure 4 Peak velocities of fast phases of vestibular nystagmus in normals (x), in patients with dyspraxia of speech (●) and in patients with pontine disorder (○). Mean and standard deviation are shown in one direction.

of these patients we were unable even to evoke either vestibular or optokinetic nystagmus by the usual stimuli, and in the remaining cases the velocity of the fast phases of vestibular nystagmus was also constantly lower than in normals (Figure 4). In these patients, although the saccades were slower than in normals, the saccades were always faster than the fast phases of nystagmus. At amplitudes of 60° the mean peak velocity of the saccades of these patients was 280° s^{-1} as compared with 500° s^{-1} for normal subjects (Figure 3). For 30° amplitude the mean velocity of the fast phases of nystagmus of these patients was 150° s^{-1} as compared with 280° s^{-1} in normal subjects (Figure 4).

The accuracy of the saccades varied among these patients; four had a decrease in accuracy and four were normal in this respect. The patient with a tumour in the IVth ventricle

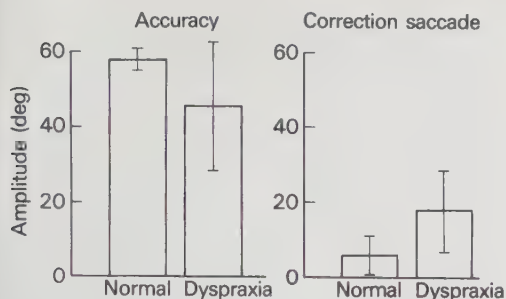


Figure 5 Accuracy of voluntary saccades at the amplitude of 60° in normal subjects and in patients with dyspraxia of speech. Mean and standard deviations are shown.

could not perform saccadic eye movements of more than 50°, but his saccades were accurate up to 40°. Figure 2 shows a recording of his saccades.

PATIENTS WITH ACUTE ENCEPHALITIS

The peak velocities of the saccades in the patients with acute encephalitis were high (Figure 6). Also the velocity of the fast phases of nystagmus in these patients was significantly higher than in normal subjects (Figure 7). The accuracy, however, was lower than in normals; in order to reach the target these patients had to use correction saccades with a higher amplitude than normal subjects (Figure 8). In contrast to patients with dyspraxia they usually used only one large correction saccade to reach the target.

PATIENTS WITH PERIPHERAL VESTIBULAR DISORDER

No differences were found in the peak saccadic velocity between normal subjects and patients with peripheral vestibular disorders (Figure 6). The peak velocity of the fast phases of nystagmus was the same as in normal subjects (Figure 7). The mean accuracy of the saccades and the mean amplitude of correction saccades were also normal (figure 8).

Statistics

A statistical analysis of the peak velocities of the saccades at amplitudes of 20° is shown in Table 2. A significant difference was found between normal subjects and patients with pontine disorders as well as between normal subjects and patients with encephalitis.

Table 3 shows the statistical analysis of the velocity of the fast phases of nystagmus at amplitudes of 20°. Patients with changes in saccadic velocity presented corresponding changes also in quick phases of nystagmus. Significant changes were present in cases with pontine disorders (decrease) and with encephalitis (increase), while quick phase velocity in patients with dyspraxia of speech and of eye movements did not differ significantly from that of normals.

Table 4 shows the statistical analysis of the accuracy of the saccades expressed as amplitude

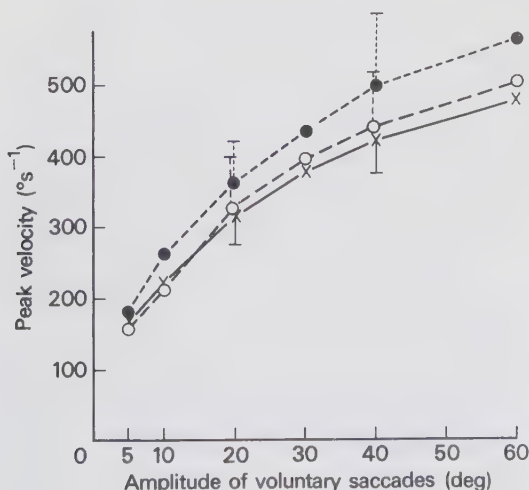


Figure 6 Peak velocities of voluntary saccades in normals (x), in patients with encephalitis (●), with vestibular disease (o). Mean and standard deviation are shown.

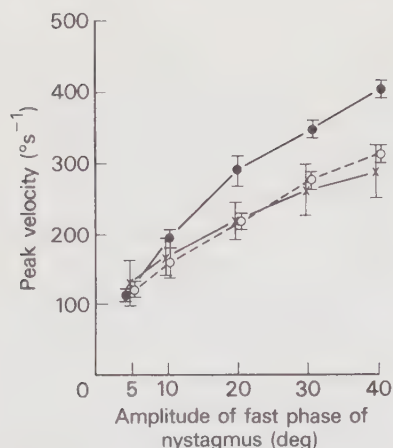


Figure 7 Peak velocities of fast phases of vestibular nystagmus in the same patients as in Figure 6. Mean and standard deviation are given. Symbols as Figure 6.

of the primary and of the correction saccades when the subjects were trying to perform saccades at the amplitude of 60° .

A significant difference in the accuracy of the movements expressed as an amplitude of the primary saccades was found between normal subjects on one hand and patients with dyspraxia or with meningo-encephalitis on the other. The difference of the amplitudes of the correcting saccades was even greater. No statistical difference could be revealed between patients with dyspraxia and meningoencephalitis with regard either to primary or to correction saccade amplitude.

Discussion

In 3 of the 4 groups of patients, distinct patterns were found in respect of the accuracy of saccades, the velocity of the saccades as well as the velocity of the quick phase of nystagmus.

PATIENTS WITH DYSPRAXIA OF SPEECH

This group was characterized by a clear-cut decrease in saccadic accuracy in combination with normal saccadic and quick phase velocity, a pattern termed 'dyspraxia of gaze' by Waltz.²⁴ The combination of dyspraxia of speech with eye-motor dyspraxia would seem to suggest the following aspects of the initiation of voluntary saccades.

The precise mechanism by which voluntary saccades are initiated in the human cortex is still obscure.²⁶ Although premotor potentials appear in the EEG of the human frontal cortex,^{2,27,28} single unit recordings in alert monkey have failed to reveal any presaccadic neuronal activity.^{1,8,9} However, several such 'presaccadic units' have been found in the visual associative cortex of the cat^{29,30} and in the posterior parietal cortex of the monkey^{3,10,31}. In the monkey these regions are activated presaccadically only if the monkey takes a special interest in the object.³

The absence of presaccadic activity in the frontal lobe of experimental animals may not apply to humans since saccadic motivation may

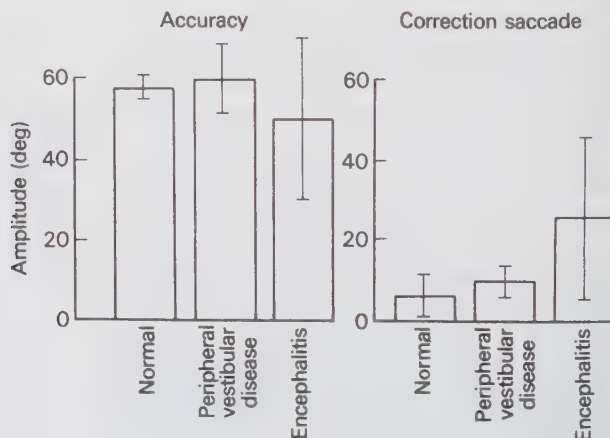


Figure 8 Accuracy of voluntary saccades at the amplitude of 60° in the same patient groups as in Figure 6. Mean and standard deviation are given.

Table 2 Difference between normals and patients in peak velocity of saccades in light, amplitude 20° (*t*-test)

Variable 1	Variable 2	<i>t</i> -value	<i>n</i>	Significance
Normal	Dyspraxia of speech	0.85	25	n.s.
Normal	Pontine disorder	4.56	26	$P < 0.001$
Normal	Encephalitis	2.32	23	$P < 0.025$
Normal	Vestibular disease	0.80	24	n.s.

Table 3 Difference between normals and patients in peak velocity of fast phase of rotatory nystagmus, amplitude 20° (*t*-test)

Variable 1	Variable 2	<i>t</i> -value	<i>n</i>	Significance
Normal	Dyspraxia of speech	1.36	25	n.s.
Normal	Pontine disorder	9.03	26	$P < 0.001$
Normal	Encephalitis	5.47	23	$P < 0.001$
Normal	Vestibular disease	1.61	24	n.s.

be different. In monkeys it is probably not possible to induce true voluntary eye movements without stimulating interest in the target.³² Consequently, existing evidence of the role of area 8 in inducing voluntary saccades is still based primarily on animal experiments.

With this as a background, the combination of speech dyspraxia and eye-motor dyspraxia may indicate that the human voluntary saccades may be initiated in the frontal pre-motor cortex. The close coupling could, however, also be understood if the neurons of the frontal cortex do not initiate saccades but serve as a feed-back control of saccades initiated elsewhere in the CNS.

Independent of whether saccades are generated or only controlled in the frontal cortex, the finding of failing eye-motor accuracy should indicate a disturbance at a cortical level in the frontal lobe. Much previous information also seems to support the idea that saccades are controlled by the cortex.³¹

When saccadic inaccuracy is encountered, however, other possibilities must also be taken into account. Little is known about the role played by the superior colliculi,¹⁴ the basal ganglia¹⁸ and the cerebellum^{33,34} in processing the cortical signal. Typically, a derangement in any of these structures causes inaccuracy in voluntary saccades. It is possible that some additional characteristics of saccades, i.e. correction or reaction time can distinguish between various levels of lesions responsible for accurate voluntary saccades.

PATIENTS WITH PONTINE DISORDERS

In this group saccadic accuracy showed a wide spread from normality to a pronounced derangement. The decrease in velocity of the rapid eye movements was a consistent finding and can be interpreted along the lines of recent electrophysiological results. Efferent pathways from the various centres for initiation of rapid eye movements are projected on to the pons and are channeled through a 'final common pathway'³⁵ to the eye motor nuclei^{7,36-39}. Eye-motor impulses converge towards the paramedian reticular formation (PPRF), where the same neurons are activated synchronously with certain positions of the eyes. Some neurons seem to be activated during pursuit eye movements and others during rapid eye movements with identical patterns during saccades and fast phases of nystagmus.⁷ A lesion at this level of neuronal convergence should be expected to reduce the velocity of saccadic eye movements as well as of the nystagmic quick phase which, in fact, in most cases did take place.

The dissociation, which was sometimes found between saccadic velocity and the velocity of the quick phase of nystagmus, can also be

Table 4 Difference between normals and patients in accuracy (amplitude of first saccadic effort in relation to target amplitude) and amplitude of correction saccades (*t*-test)

Variable 1	Variable 2	<i>n</i>	Accuracy		Correction saccade	
			<i>t</i> -value	significance	<i>t</i> -value	Significance
Normal	Dyspraxia of speech	25	2.83	$P < 0.05$	4.28	$P < 0.001$
Normal	Encephalitis	23	2.11	$P < 0.05$	3.95	$P < 0.001$
Normal	Vestibular disease	24	0.54	n.s.	1.11	n.s.
Dyspraxia of speech	Encephalitis	10	0.23	n.s.	0.32	n.s.

explained along these lines. Tentatively, in such a case the lesion may selectively affect only one type of eye-motor impulses and this at a level before which the eye-motor unit in PPRF is reached.⁴⁰

PATIENTS WITH ENCEPHALITIS

The inaccuracy of eye movements in these cases may be due to a cortical and/or cerebellar dysfunction. The increase in velocity of the rapid eye movements, saccades as well as quick phases of nystagmus, may be due to loss of the inhibition normally exerted by these structures on the pontine centres.

PATIENTS WITH PERIPHERAL VESTIBULAR DISORDERS

The patients with peripheral vestibular disorders did not show any significant changes in any of the tested parameter. Therefore, this kind of analysis is not useful for discriminating between different types of peripheral vertigo. However, it should be helpful in discriminating between vertigo of peripheral or central origin as well as between various types of central disturbances causing vertigo, unsteadiness or dizziness.

When the average velocity of any type of rapid eye movements was disturbed, this was reflected in all three types, i.e. the saccades and in the quick phases of vestibular and optokinetic nystagmus as is also seen in Table 4. Consequently for clinical purpose it may be sufficient to analyse only one type of rapid eye movement. However, as was mentioned above a dissociation is sometimes seen between the velocity of saccades and of quick phases of nystagmus. In such cases further information is obtained by determining saccadic as well as quick phase velocity.

The determination of accuracy as well as velocity of voluntary saccades together with the velocity of fast phases of nystagmus may thus provide valuable tools for clinical diagnosis.

It must, however, be stressed that various factors severely influence the velocity of rapid eye movements. Thus the saccadic velocity seems to depend on visual conditions, on

alertness of the subject, on prolonged testing, on drugs and on many other factors discussed in a previous paper.²³ Under standardized condition it is, however, possible to determine the velocity of rapid eye movements with acceptable inter- and intra- individual variations.^{23,41,42} Furthermore, the temporal profile of the lesion and the ability of the central nervous system to compensate for the deficit has also to be taken into account when evaluating rapid eye movements.

As all routine nystagmographic tests provide nystagmus beats as well as saccadic eye movements for calibration, any nystagmogram will allow the type of analysis suggested here. A proper evaluation will be possible, however, only if the paper is fed at a sufficiently high speed, preferably at about 100 mm s^{-1} .

Thus, if we consider these restrictions and modifications, this type of evaluation of rapid eye movements seems promising for deducing further information about the CNS from eye-motor disturbances.

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QUANTIFICATION OF TRACKING EYE MOVEMENTS IN NORMAL SUBJECTS

Lucyna Schalén

Department of Otorhinolaryngology, University Hospital, Lund, Sweden

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Abstract. Voluntary tracking eye movements were analysed in 20 normal subjects whose gaze was fixed on a visual target moving at six different constant velocities between 10° s^{-1} and 60° s^{-1} . Tracking ability was quantified according to four parameters. The mean value and dispersion of each parameter at each velocity were determined. *The maximum velocity gain of smooth pursuit* was, on average, 0.98–0.75, gradually diminishing with increasing target velocities of 10 – 60° s^{-1} . *Amplitude of smooth pursuit* decreased gradually and was replaced by superimposed saccades at increasing target velocities. *Saccades* with amplitudes of 3 – 10° were present at all target velocities, while those greater than 10° occurred mostly at target velocities above 30° s^{-1} . *Square waves* were rare but equally frequent at all target velocities and seemed to occur randomly during tracking eye movements.

An additional group of 9 subjects was investigated twice. Mean values of maximum velocity gain, of amplitude of smooth pursuit and of frequency of superimposed saccades were higher on the second occasion, probably reflecting the effect of learning.

Co-operation and interaction of the smooth pursuit and the saccadic subsystems to produce the voluntary tracking were discussed.

During voluntary tracking eye movements the image of a visual target is kept on the fovea despite movements of the body, the head, or of the target. The accuracy of tracking has been analysed mostly with respect to velocity gain, i.e. the ratio of eye velocity/velocity of the target. It has been shown that the gain is close to 1.00 at low target velocities (Baloh et al., 1976; Kowler et al., 1978; Sharpe et al., 1979). However, the amplitude of the pursuit eye movements in relation to the movement of the target has not received much attention. Few efforts have been made to quantify the superimposed saccades, i.e. those rapid eye movements which fix the fovea again on the target in case of a foveal image slip during

tracking. Although the phenomenon has been studied previously (Westheimer, 1954; Brown, 1972) quantitative analyses of saccadic corrections with respect to different target velocities have rarely been performed and in those instances the analysis was concentrated on saccadic frequencies but not on saccadic amplitudes (Sharpe & Sylvester, 1978) or else the testing applied to only one target velocity (Hartje et al., 1978).

Square waves interfering with smooth pursuit eye movements have mostly been interpreted as indicative of a pathological condition (Dell'Osso et al., 1975; Feldon & Langston, 1977) or as resulting from the lack of visual control of eye movements (Kornhuber, 1974). Only a qualitative description of square waves during fixation on a moving target in healthy subjects has so far been published (Jung & Kornhuber, 1974).

The aim of the present investigation was to analyse the variation in voluntary tracking eye movements at different target velocities, with special reference to velocity gain, smooth pursuit amplitude, saccadic corrections and square waves.

MATERIAL AND METHODS

Tracking movements of the eyes were studied in 20 volunteers, 10 males and 10 females of mean age 49.9 (range 22–70) years.

An additional group of 9 volunteers was examined on two separate occasions in order to study intra-individual variation. This group

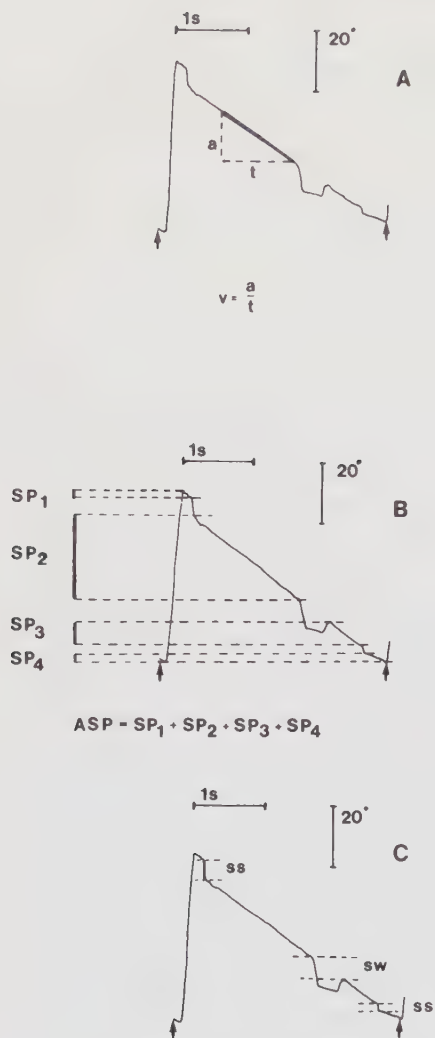


Fig. 1. Evaluation of EOG recordings of voluntary tracking eye movements in a normal subject at target velocity of 10° s^{-1} . Arrows indicate extreme positions of the visual target. A: saccade to the right brings the eyes on the target. Subsequent tracking to the left consists of smooth pursuits (SP), superimposed saccades (SS) and square waves (SW). B: Amplitude of smooth pursuit (ASP). C: Superimposed saccades and square waves.

consisted of 3 women and 6 men, aged 22–34 (mean 25.3) years.

The subjects had no previous history of neurological or otological disturbances and no signs of pathology were found at the otoneurological examination (Henriksson et al., 1972). No drugs were allowed within 48 hours of the test (Rashbass, 1961).

The visual target was a spot of light, 30 mm in diameter, projected on a screen 160 cm from the subject. The target was driven in predictable ramps of 60° amplitude at the six velocities of 10° , 20° , 30° , 40° , 50° , 60° s^{-1} . The target could travel in both directions. As soon as the target disappeared in one direction, a new one became visible on the opposite edge of the screen. Extreme positions of the target on the screen were detected by phototransistors and recorded.

The subject was seated in a chair, with the head fixed by occipital supports. The movements of the eyes could be monitored during the test with an infrared-operated TV camera. The subject was instructed to pay attention to the target and to follow it as accurately as possible, avoiding blinking. Verbal contact was maintained throughout the procedure in order to keep the attention on the visual task.

Each test procedure was started by a tracking to the right at the lowest target velocity. Five consecutive trackings were recorded at each velocity. The procedure was subsequently repeated to the left.

Eye movements were recorded with a d.c. electro-oculographic technique, using commercial electrodes and jelly (Medicotest®). Horizontal eye movements were recorded binocularly and monocularly in the vertical plane (Henriksson et al., 1972; Gay et al., 1974). Recordings were displayed by a jet-inewriter (Mingograph M-81, Siemens-Elema, Stockholm, Sweden) with an upper cut-off frequency of 15 Hz. The recording paper was fed at a velocity of 25 mm s^{-1} allowing an accuracy of time events of 40 ms, corresponding to 1 mm in the recording. The recordings permitted the resolution of eye position to an accuracy of 1° , corresponding to 1 mm on the recording. Calibration of eye movements was taken from voluntary saccades 20° and 60° to the left and to the right.

The following parameters were evaluated:

The maximum velocity gain of smooth pursuit eye movement (MVSP gain) was calculated from the ratio maximum velocity of smooth

Table I. *Tracking eye movements in 20 normal subjects*

Means of the individual means and standard deviations (in parentheses) of the tested parameters during tracking to the right at different target velocities

	Target velocity (deg s ⁻¹)					
	10	20	30	40	50	60
Maximal velocity gain	0.95 (0.10)	0.98 (0.12)	0.94 (0.12)	0.92 (0.15)	0.86 (0.14)	0.75 (0.21)
Total tracking amplitude, in deg.	60.0 (7.7)	57.6 (8.9)	53.0 (11.2)	52.3 (9.6)	46.3 (12.4)	41.6 (11.3)
Amplitude of smooth pursuit, in deg.	52.8 (10.0)	51.1 (9.7)	45.8 (11.4)	44.5 (9.9)	36.6 (10.5)	27.2 (8.3)
Superimposed saccades frequency, in 60s of testing						
3–10°	6 (5)	11 (11)	20 (17)	33 (23)	35 (28)	34 (23)
11–20°	0	3 (5)	4 (4)	9 (14)	16 (14)	18 (20)
21°–>	0	0	1 (4)	3 (5)	4 (8)	11 (20)
Square waves frequency, in 60 s of testing	6 (10)	5 (3)	4 (8)	1 (3)	6 (6)	2 (8)

pursuit eye movement/target velocity. The maximum velocity of smooth pursuit eye movement (MVSP) was registered after an initial velocity adaptation period of 500 ms and calculated from a part of the smooth pursuit eye movement which lasted at least 250 ms, i.e. the shortest time for the visual perception of the movement (Yarbus, 1967) (Fig. 1a).

The amplitude of smooth pursuit eye movement (ASP) was defined as the sum of all smooth pursuit eye movements in each tracking after excluding saccades and square waves (Fig. 1b).

The total tracking amplitude (TA) was equal to the amplitude of returning saccades.

The superimposed saccades (SS) were classified according to the amplitudes into three groups: 3–10°, 11–20°, and 21° or more (Fig. 1c). SS smaller than 3° were not included.

The square waves (SW) were defined as two saccades directed counter to each other and having an interval of relative standstill (Fig. 1c).

Measurements were made manually and calculations by computer (Univac 11-08-EXC-8). The mean value and standard deviation of individual results for each parameter were based on five consecutive trackings at each velocity and in each direction. Altogether 1 200

trackings were recorded in 20 normal subjects; 13 trackings were excluded for technical reasons.

RESULTS

The mean value and dispersion of each parameter during voluntary tracking eye movements to the right in 20 normal subjects are given in Table I.

The maximum velocity gain of smooth pursuit eye movement (MVSP gain)

The eyes followed the target with exact velocity (MVSP gain = 1.00) in only 7.6% of all trackings.

The mean MVSP gain was close to 1.00 at target velocities of 10–40° s⁻¹ and occasionally at target velocities of 50° s⁻¹ and 60° s⁻¹ but as a rule the MVSP gain decreased with increasing target velocity (Fig. 2). Slight MVSP gain overshoots, i.e. gains of 1.01–1.30, were found in 22% of the trackings, at both low and at high target velocities. In most of the trackings (70.4%), however, the eyes lagged behind the visual target. The interindividual variation increased with increasing stimulus velocity. The variation coefficient was 15% at target velocities of 10–40° s⁻¹. As the target accelerated, the variation coefficient could exceed 30%. The intra-individual range

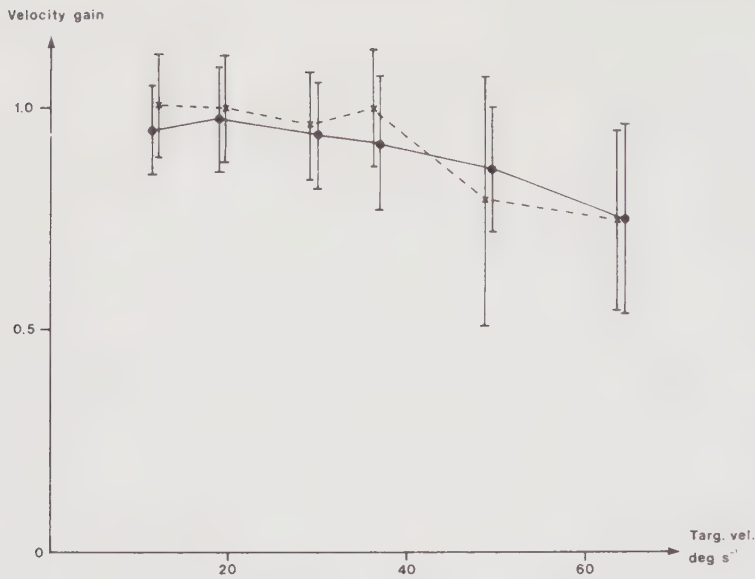


Fig. 2. Maximum velocity gain of smooth pursuit at different velocities of the visual target. Means of individual means $\pm$ S.D. in 20 normal subjects during tracking to the right (—) and to the left (- -).

of variation was also velocity dependent; up to a target velocity of 40° s^{-1} the variation coefficient between gains in the separate trackings was less than 10% in the majority of subjects. At a target velocity of 40° s^{-1} or higher, the variation of MVSP gain increased abruptly in most subjects and often exceeded 30%.

No statistically significant difference in MVSP gain between tracking to the right and to the left could be found (*t*-test for unpaired data, $p > 0.05$).

Amplitude of smooth pursuit eye movements (ASP)

The total tracking amplitude (TA) gradually decreased in inverse proportion to the increasing velocity of the visual target, corresponding to the decrease in amplitude of the returning saccade, according to the construction of the test. Parallel with the increase in target velocity, there was a percentage decrease in the amplitude of smooth pursuit. This decrease was of the same range at target velocities of

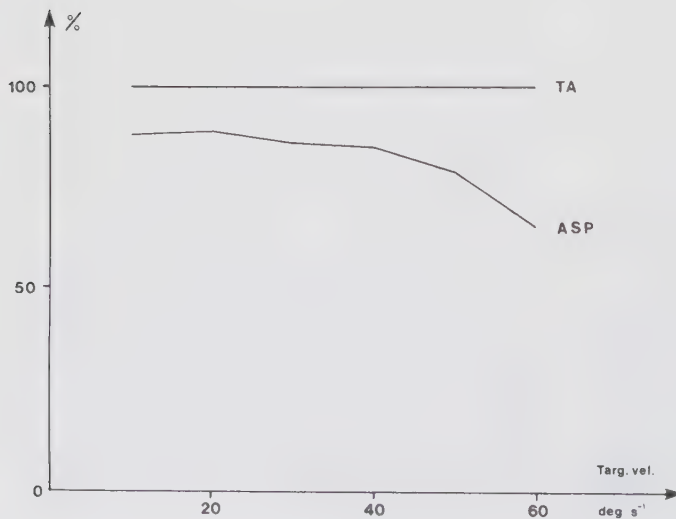


Fig. 3. Amplitude of smooth pursuit (ASP) as percentage of total tracking amplitude (TA) at increasing velocities of the visual target during voluntary tracking eye movements.

SUPERIMPOSED SACCADES

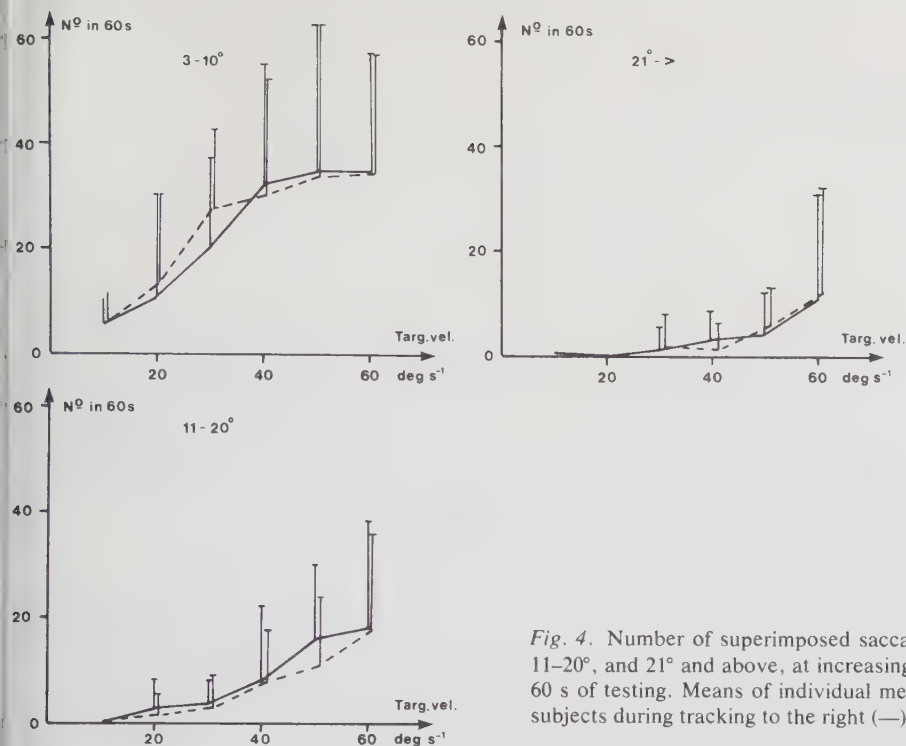


Fig. 4. Number of superimposed saccades of amplitudes 3–10°, 11–20°, and 21° and above, at increasing target velocities, during 60 s of testing. Means of individual means $\pm$ S.D. in 20 normal subjects during tracking to the right (—) and to the left (---).

10–40° s^{-1} , while above the target velocity 40° s^{-1} the percentage decrease in smooth pursuit was more abrupt (Fig. 3), to favour the saccadic mode of tracking. No significant directional difference in ASP could be found (t -test for unpaired data $p > 0.05$).

Superimposed saccades (SS)

Superimposed saccades were present in most subjects—on the average, 0.9/tracking. In 3 of the subjects, however, the saccade-free trackings were recorded. The majority of saccades, 80%, belonged to the smallest amplitude category: 3–10°. 15% of SS were in the range 11–20° and only 5% were larger than 20°. The frequency and amplitude of SS were related to the velocity of the target: saccades of 3–10° increased appreciably with increasing target velocity. Saccades of 11–20° became more common at target velocities of 30° s^{-1} and above and, finally, saccades larger than 20° appeared only at velocities of 30° s^{-1} and

above (Fig. 4). There was no statistically significant difference in frequency of SS to the right or to the left (t -test for unpaired data $p > 0.05$).

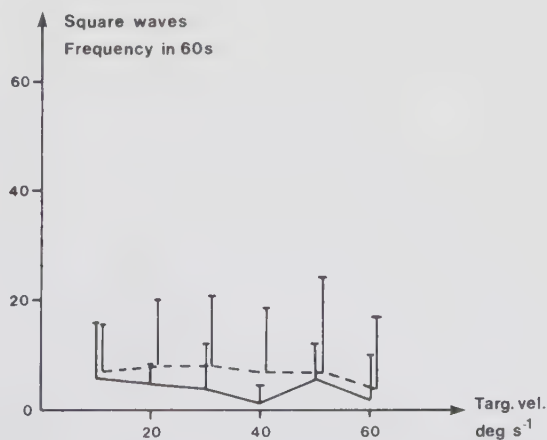


Fig. 5. Frequency of square waves at different target velocities during 60 s of testing. Means of individual means $\pm$ S.D. in 20 normal subjects during tracking to the right (—) and to the left (---).

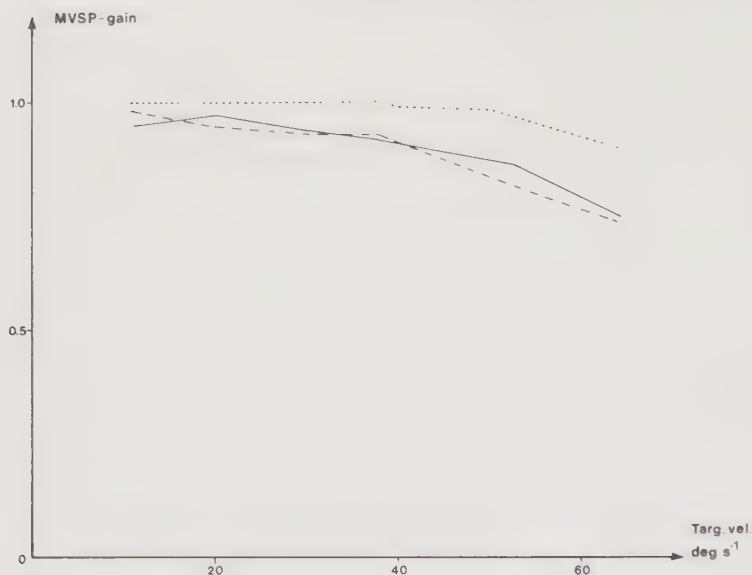


Fig. 6. Effects of repeated testing on maximum velocity gain of smooth pursuit (MVSP gain). Means of individual means in 9 subjects investigated twice were higher on the repeated testing (···) compared to the first occasion (---). The reference group (—) of 20 normal subjects was investigated only once.

Additional testing group

Nine additional subjects were tested twice at intervals of 1–7 days. On the first occasion there was no statistically significant difference in the mean results compared with the main group of 20 subjects with respect to MVSP gain, ASP, SS and SW. However, on the second occasion MVSP gain and ASP (Figs. 6 and

7) were significantly higher (*t*-test for paired samples, $p < 0.05$) at all target velocities except 60° s^{-1} . Also there were more SS of $3\text{--}20^\circ$ during the second test, while the frequency of SS with amplitudes of 21° and above remained constant (Fig. 8). There was no statistically significant difference in the frequency of SW during the first vs. the second investigation.

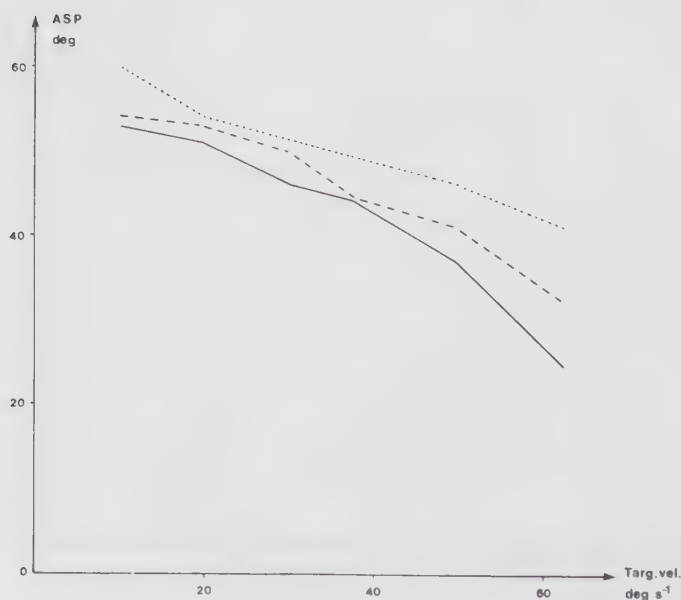


Fig. 7. Effects of repeated testing on amplitude of smooth pursuit. Means of individual means in 9 subjects investigated on the first (---) compared with the second (···) occasion, and to the reference group of 20 normal subjects (—) investigated once.

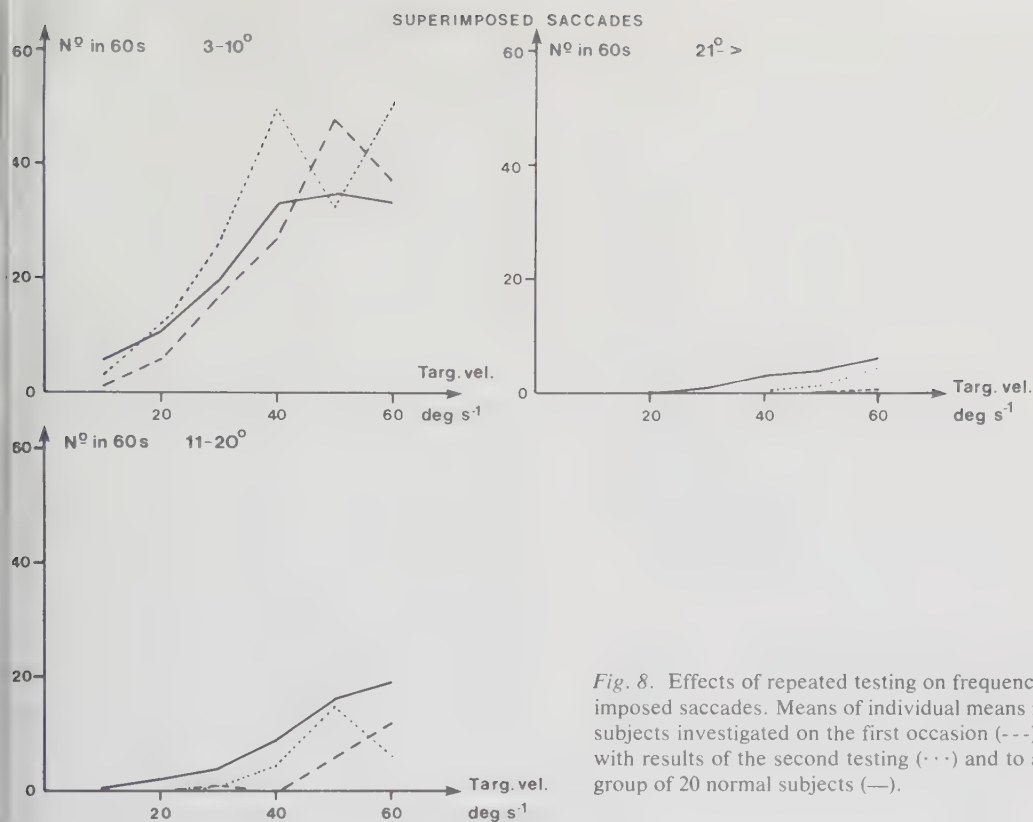


Fig. 8. Effects of repeated testing on frequency of superimposed saccades. Means of individual means in 9 normal subjects investigated on the first occasion (---) compared with results of the second testing (···) and to a reference group of 20 normal subjects (—).

DISCUSSION

Adaptation of eye velocity to velocity of the target during smooth pursuit

Eye velocity, even during a single tracking, seldom remains constant and shows considerable variation (Westheimer, 1954; Brown, 1972; Williams & Fender, 1979). Different arbitrary parameters have therefore been used to express eye velocity during smooth pursuit, e.g. mode eye velocity (Baloh et al., 1976) and average velocity (Brown, 1972; Kowler et al., 1978). In the present paper the maximum velocity of smooth pursuit was chosen in order to limit sources of error due to possible temporary slackening of attention. The mean values of maximum velocity gain of smooth pursuit (MVSP gain) at different target velocities were comparable to results obtained by other authors (Baloh et al., 1976; Sharpe et al., 1979). In agreement with the earlier investigations

the mean MVSP gain decreased in inverse proportion to increasing target velocities.

The present material indicates the limited ability of the eyes to match velocity of the target: the ideal gain was achieved in only 7.6% of the trackings, in 70.4% eye velocity was lower than that of the target and velocity overshoots arose in 22% of the trackings. Williams & Fender (1979) proposed recently that the poor velocity adaptation and velocity overshoots derive from the sloppy character of the smooth pursuit control system. It has also been shown that both high velocity gains and overshoots arise mostly when tracking targets move in a predictable manner (Stark et al., 1962; Kowler et al., 1978, Sharpe & Sylvester, 1978). As the present test used predictive tracking, the velocity overshoots might be, at least partly, a result of overcorrection of anticipated velocity error.

Visual perception of target during tracking eye movements

As each subject can deliberately start or interrupt tracking eye movements at any moment instantaneously, it was considered of interest to monitor the overall excursion of the eyes during tracking (TA) as well as the amplitude of smooth pursuit (ASP). Since perception of the moving object is possible only during pursuit (Yarbus, 1967) the ASP is expected to reflect the extent to which the target was seen during the tracking. Hence, the target can be visually monitored to roughly the same degree at target velocities ranging from 10° to 40° s^{-1} (during 85–89% of each tracking), whereas continuous visual control of the moving target falls off abruptly at higher target velocities.

Characteristics and origin of square waves

In contrast to the observed increase in superimposed saccades, the number of square waves remained fairly constant at different target velocities (cf. Figs. 4 and 5). Another difference between SS and SW was that saccades were always directed 'with' the target, whereas primary saccades in the SW could occasionally turn the eyes in the opposite direction. These observations suggest that saccadic components in SW are not produced in order to correct eye velocity or any other constant error of smooth pursuit. The eyes would appear to jump randomly during the SW, possibly reflecting a random activity of pontine neurons in normal subjects.

Co-operation of smooth pursuit and saccadic systems in matching eye- and target position

When one's interest focuses on a moving target the eye motor system receives the information of target's position and velocity. Both the smooth pursuit and the saccadic subsystems of eye movement control are activated. Continuous visual feed-back is necessary to fix the eyes on the target during smooth pursuit (Robinson, 1965). The present results indicate a velocity of 40° s^{-1} as the limit for optimal smooth pursuit. Above this range, along with

the decrease of visual perception, the velocity gain was seldom close to 1.00 and an abrupt increase in inter- and intra-individual variations was observed (Fig. 2). At the same time saccades covered more than 20% of total tracking amplitude (Fig. 3). Tracking entirely free from saccades could also occur, as observed in 3 of the subjects, indicating that the smooth pursuit system can function above optimal velocity limits. This type of tracking may be explained by a high degree of attention and motivation (Kommerell & Täumer, 1972; Troost et al., 1972). In most of the subjects, however, an increase in saccadic frequency was observed with increasing target velocities, thus supporting the theory that the saccadic system has to be activated when the smooth pursuit system itself cannot adequately control the position of the eyes (Williams & Fender, 1979).

The saccades produced in order to catch up with the target are believed to be induced either by retinal information on change in target position (Rashbass, 1961) or else extraretinally, due to anticipated positional error of the eyes (Zee et al., 1974).

In the present material most of the saccades seemed to be induced and controlled by retinal mechanisms as saccadic amplitudes were related to the velocity of the target. Moreover, the frequency of saccades allowed time courses sufficient for the retinal signal processing (see Monnier, 1952). However, occasionally saccades could be very frequent, appearing at intervals shorter than the minimal 150 ms (Stark et al., 1962), especially at target velocities of 50° and 60° s^{-1} . Hence, the anticipatory mechanism of saccadic control must be considered. It is proposed that both the retinal and the extraretinal mechanism of control of superimposed saccades are functioning during voluntary tracking. The anticipatory mechanisms seem to be activated mostly at high velocities of the visual target.

Effects of repeating the test

A significant change in the mean results of the 9 subjects investigated twice was found on

repeated testing, i.e. higher MVSP gains and ASP as well as a higher frequency of saccades of 3–20°. This could be interpreted as an effect of training in motivated subjects (de Weese Puckett & Steinman, 1969). The change in eye movement function seems to apply to both velocity matching and correction of positional errors.

CONCLUSIONS

In the present work, an effort to quantify voluntary tracking has been made by applying four parameters: maximum velocity gain of smooth pursuit (MVSP gain), amplitude of smooth pursuit (ASP), frequency of superimposed saccades (SS) of different amplitude, and frequency of square waves (SW).

An adequate following, viz. an MVSP gain close to 1.00, an ASP close to overall tracking amplitude, and a limited number of SS and SWs were recorded mainly at low target velocities. A target velocity of 40° s⁻¹ appears to be the limit for optimal functioning of the smooth pursuit system. At higher velocities the visual control of the target's movement decreases and the saccadic system will be activated to an increasing extent. Superimposed saccades at target velocities above 40° s⁻¹ seem to be triggered to some extent due to extraretinal control of targets position.

Square waves appear to occur randomly and without any corrective function during the tracking eye movements.

Training activates the smooth pursuit system as well as the saccadic system to perform the voluntary tracking with desired precision.

A forthcoming study will elucidate the extent to which the different parameters of tracking eye movements are affected in patients suffering from labyrinthine and central nervous system disorders.

ZUSAMMENFASSUNG

Vom Willen abhängige und verfolgende Augenbewegungen wurden bei 20 normalen Personen analysiert, welche mit dem Blick einem beweglichen optischen

Stimulus bei sechs verschiedenen, konstanten Geschwindigkeiten zwischen 10 und 60° s⁻¹ folgten. Die Fähigkeit zur verfolgenden Augenbewegung wurde mit Hilfe von vier Parametern quantifiziert. Der Durchschnittswert und die Ausbreitung jedes Parameters wurde bei jeder Geschwindigkeit bestimmt. „Gain“ der maximalen Geschwindigkeit von „smooth pursuit“ betrug durchschnittlich 0,98–0,75 und verminderte sich stufenweise mit zunehmender Geschwindigkeit des optischen Gegenstandes. Die Amplitude von „smooth pursuit“ reduzierte sich ebenfalls sukzessiv, wenn die Geschwindigkeit des Gegenstandes zunahm und „smooth pursuit“ durch immer frequentere Saccaden ersetzt wurde. Saccaden mit Amplituden von 3–10° kamen bei allen Geschwindigkeiten vor, während Saccaden größer als 10° oft bei Geschwindigkeiten über 30° s⁻¹ auftraten. Square waves waren selten, erschienen aber mit der gleichen Frequenz, unabhängig von der Geschwindigkeit des Gegenstandes und sie waren anscheinend zufällig ausgelöst worden.

Eine zweite Gruppe von neun Personen wurde bei zwei separaten Gelegenheiten untersucht. Die Durchschnittswerte des „gain“ der maximalen Geschwindigkeit von „smooth pursuit“, die Amplitude von „smooth pursuit“ sowie die Frequenz von Saccaden waren höher bei der zweiten Untersuchung, wahrscheinlich als Wirkung des Einlernens.

Die Kooperation zwischen den beiden Systemen der Augenbewegung – saccadische und „smooth pursuit“ – während der vom Willen abhängigen verfolgenden Augenbewegungen wurde diskutiert.

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- Lucyna Schalén, M.D.
Department of Otorhinolaryngology
University Hospital of Lund
S-221 85 Lund
Sweden

QUANTIFICATION OF TRACKING EYE MOVEMENTS IN PATIENTS
WITH NEUROLOGICAL DISORDERS

By

L.Schalén, N.G.Henriksson and I.Pyykkö

Department of Otorhinolaryngology, University Hospital,
Lund, Sweden

ABSTRACT

Tracking eye movements were studied in 20 healthy subjects and in 24 patients with vestibular neuronitis, disorders within the frontal cortex, the cerebellum or the brain stem. The tracking tests were performed at six different target velocities between $10-60^{\circ}\text{s}^{-1}$ and different parameters were evaluated. The maximum velocity gain of smooth pursuit was normal in the group with vestibular neuronitis but reduced in the groups with disorders within the frontal cortex, cerebellum and the brain stem. The total amplitude of tracking and amplitude of smooth pursuit were normal in the groups with vestibular neuronitis and frontal cortical disorders while in the groups with cerebellar and brain stem disorders it was reduced. The frequency of superimposed saccades with amplitude range of $3-10^{\circ}$ was significantly increased in all groups; however, in the group with vestibular neuronitis the increase was significant only during tracking in the direction of spontaneous nystagmus. The number of superimposed saccades with amplitude range of $11-20^{\circ}$ was significantly increased only in the group with cerebellar disorders. The frequency of square waves was not significantly increased in any group.

Quantitative analysis of tracking eye movements seems to be a valuable aid for evaluation of site of lesions in the central nervous system.

Tracking eye movements consist of two components: smooth pursuits and superimposed saccades. The two types of eye movements cooperate in order to control the position of the eyes in relation to the moving target. Functional disturbances at any level of the central nervous system (CNS) can alter the relationship between the smooth pursuits and the saccades: the saccadic component would then increase and smooth pursuit decrease. The altered pattern of tracking in such cases has not been assumed to exhibit any specific features

with respect to the localization of the disturbance in the CNS (Gay et al., 1974; Baloh & Honrubia, 1979).

Attempts to quantify tracking eye movements have, however, revealed that saccadition may be significantly increased in patients with organic cerebral disorders, vis-à-vis functional cerebral disorders (Hartje & Kerschensteiner, 1973). Reduced velocity of smooth pursuit has been reported in patients with various cerebral cortical lesions (Troost et al., 1972; Baloh et al., 1977) and in cerebello-pontine diseases (Zee et al., 1976; Baloh et al., 1977).

A relatively wide range of normal variation in the methods used has made it difficult to evaluate the results of tracking in clinical cases. Recently it was shown by one of the present authors (Schalén, 1980) that individual ability to track could be quantified with a variety of parameters: maximum velocity gain of smooth pursuit, amplitude of tracking and of smooth pursuit, frequency and amplitudes of superimposed saccades, and frequency of square waves.

The aim of the present study was to evaluate whether organic disorders at certain sites in the CNS might cause specific changes in the pattern of tracking eye movements. For this purpose the four above mentioned parameters of the tracking test were studied in four groups of patients with organic lesions in different parts of the CNS.

MATERIAL AND METHODS

Subjects

Twenty-four consecutive patients with disorders in either the peripheral vestibular organ, frontal cortex, cerebellum or brain stem were investigated. Each of the four diagnostic groups consisted of 6 subjects. For comparison values from 20 normal subjects of mean age 49.9 (range 22-70) years were used (Schalén, 1980).

Vestibular neuronitis, determined according to Dix & Hallpike (1952) was found in 3 males and 3 females of mean age 57.7 (range 40-69). They displayed unilateral canal paresis without any other neurological signs. The eye movements were recorded 6-30 days after the onset of acute vertigo.

Frontal cortical disorders were diagnosed in 6 males, of mean age 61.8 (range 38-73) years. All patients had dyspraxia of speech indicating that the damage involved the left cerebral cortex including the frontal area 8 (Dahlen et al., 1980). The dyspraxia occurred due to vascular stroke in 5 patients; one patient suffered from a degenerative process of the Niemann-Pick type. The eye movements were recorded 2-48 months after the onset of the cerebral symptoms.

Cerebellar disorders were identified in 4 males and 2 females of mean age 46 (range 12-72) years. Two patients had tumours, two degenerative cerebellar disease, one suffered from acute encephalitis, and one had a vascular infarction. Eye movements were examined 3 weeks to 10 years after the onset of clinical symptoms.

Brain stem disorders were found in 2 males and 4 females of mean age 66 (range 46-72) years. The etiology was vascular infarction in 4 and progressive supranuclear palsy in 2 of the subjects. The patients were

examined one week to 6 months after the debut of symptoms.

All patients were quite cooperative. Any CNS-influencing drugs were discontinued at least 48 hours prior to the investigation.

Eye movement recordings

The subject was seated in a chair and the head was fixed with occipital supports. The movements of the eyes were monitored with an infrared TV camera. Eye movements were recorded with a d.c. electro-oculography and displayed by an ink-jet writer (Mingograph M 81, Siemens Elema, Stockholm, Sweden) with an upper cut-off frequency of 15 Hz. Mingograph paper was fed at a speed of 25 mm s^{-1} , allowing the resolution of time events to 40 ms (corresponding to 1 mm in recording). The accuracy of eye position was 1° (corresponding to 1 mm in the recordings). Calibration of eye movements was taken from voluntary saccades of 20° and 60° .

The tracking test

Before the test each subject was instructed to track a visual target as accurately as possible. Verbal contact was maintained throughout the procedure in order to sustain attention.

The visual target was a spot of light, 30 mm in diameter, projected onto a screen 160 cm from the subject. The target moved horizontally at constant velocities of 10, 20, 30, 40, 50 and 60°s^{-1} and its excursion covered 60° of amplitude. As soon as the target disappeared in one direction, a new one became visible on the opposite edge of the screen. Thus, sequences of tracking eye movements in the same direction as the target and returning saccades in opposite direction were induced. Tracking eye movements consisted of smooth pursuits, superimposed saccades and square waves.

The following parameters were evaluated:

The maximum velocity gain of smooth pursuit calculated from ratio maximum velocity of smooth pursuit eye movement/target velocity. The maximum velocity of smooth pursuit eye movement was registered after an initial velocity adaptation period of 500 ms and calculated from a part of smooth pursuit eye movement which lasted at least 250 ms.

The amplitude of smooth pursuit eye movement, i.e. the sum of amplitudes of all smooth pursuit eye movements during every tracking and the total amplitude of tracking, i.e. the sum of amplitudes of smooth pursuits, superimposed saccades and square waves.

The frequency and amplitude of superimposed saccades. Superimposed saccades were classified in different amplitude classes of $3-10^{\circ}$, $11-20^{\circ}$ and 21° and above.

The frequency of square waves, i.e. two saccades directed counter to each other and having an interval of relative stand still.

All parameters were calculated in five consecutive trackings at each velocity and in both directions. The mean of the five responses was given as individual value for each subject at every velocity and direction. Depending on the velocity of the target the actual duration of tracking varied (6, 3, 2, 1.5, 1.2 and 1 sec at the respective target velocities of 10, 20, 30, 40, 50 and 60°s^{-1}). The frequencies of saccades and of square waves were therefore extrapolated to a tracking time of 60 sec at each velocity and direction. The means of the individual responses within each group of patients were calculated and compared with the mean values in normal subjects. Student's t-test was used for statistical calculations.

RESULTS

Vestibular neuronitis

The mean maximum velocity gain of smooth pursuit at different target velocities in the vestibular neuronitis group was somewhat lower than in normal subjects (Fig. 1), but the differences were not statistically significant. The total amplitude of tracking was normal. The amplitude of smooth pursuit was slightly reduced and superimposed saccades of $3-10^{\circ}$ occurred more often than in normals (Fig. 1). The increase in saccadic frequency was statistically significant for tracking directed concordantly with the fast phases of spontaneous nystagmus ($p < 0.05$), but only at the target velocity of 10°s^{-1} . The superimposed saccades with amplitudes larger than 10° and the square waves occurred with normal frequency.

Frontal cortical disorders

One patient in the dyspraxia group was able to track within the normal limits at all target velocities. In the whole group the curve displaying the mean values of maximum velocity gain of smooth pursuit was lower than in normal subjects (Fig. 2). The difference was statistically significant ($p < 0.05$) at target velocities of 40°s^{-1} and above. The total amplitude of tracking and amplitude of smooth pursuit were only slightly reduced, but without statistical significance. Superimposed saccades with amplitudes of $3-10^{\circ}$ occurred significantly more frequently than in normal subjects ($p < 0.05$) (Fig. 2). However, saccades with amplitudes larger than 10° and square waves occurred with normal frequencies (Figs. 5 and 6).

Cerebellar disorders

In patients with cerebellar disorders the ability to track showed a wide range of variation, from slightly

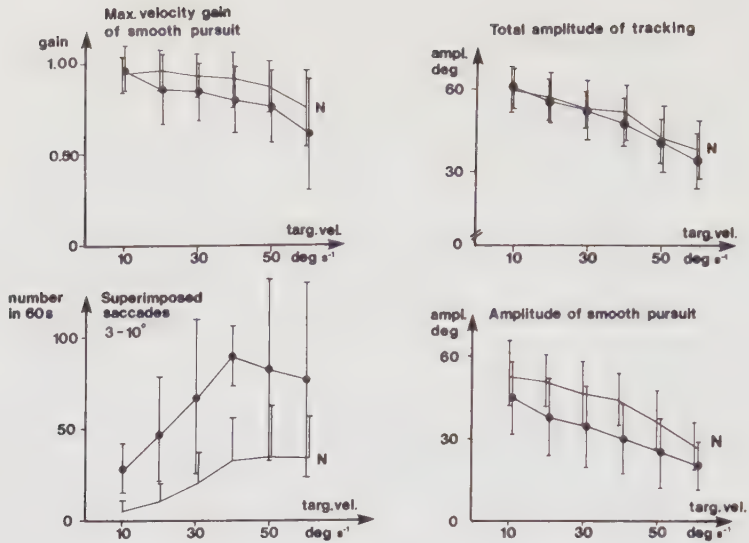


Fig. 1. Four parameters of tracking eye movements in patients with vestibular neuronitis. Means and standard deviations are given for different velocities of the visual target in 6 patients. Reference curve (N) displays means and standard deviations in 20 healthy subjects.

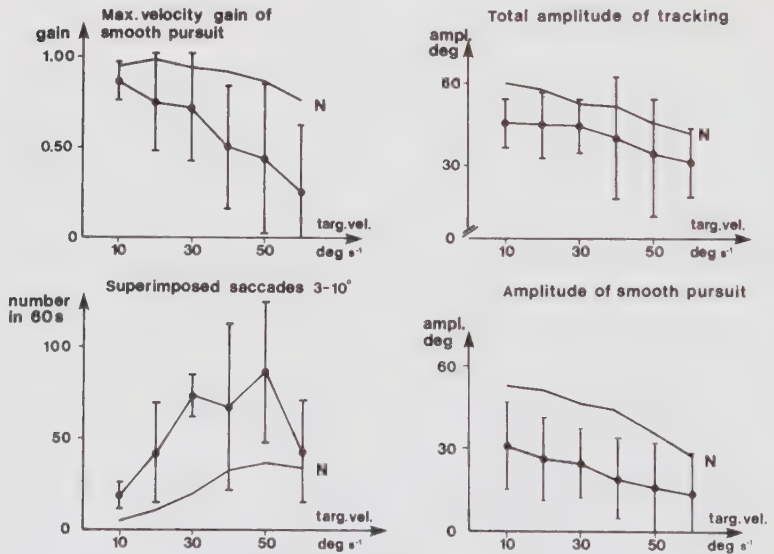


Fig. 2. Four parameters of tracking eye movements in patients with frontal cortical disorders. Means and standard deviations are given for different velocities of the visual target in 6 patients. Reference curve (N) displays means in 20 healthy subjects.

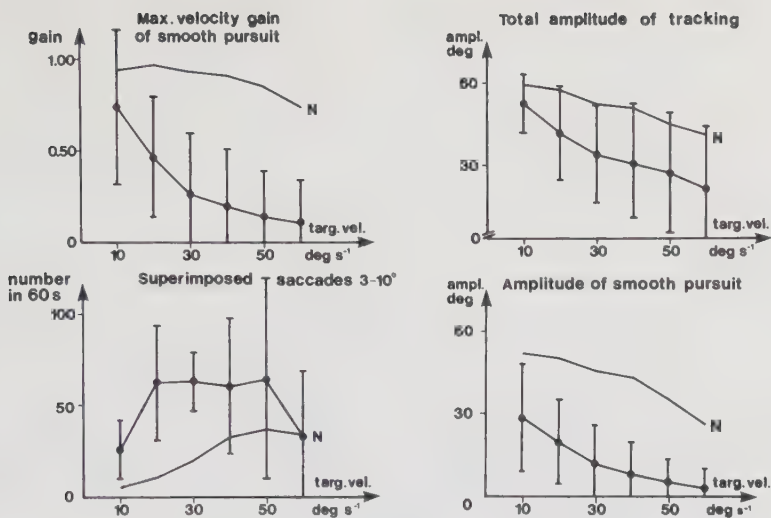


Fig. 3. Four parameters of tracking eye movements in patients with cerebellar disorders. Means and standard deviations are given for different velocities of the visual target in 6 patients. Reference curve (N) displays means in 20 healthy subjects.

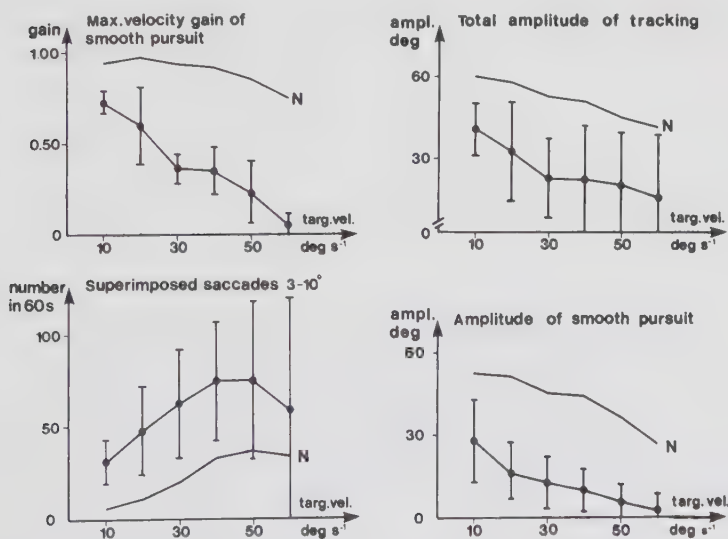


Fig. 4. Four parameters of tracking eye movements in patients with brain stem disorders. Means and standard deviations are given for different velocities of the visual target in 6 patients. Reference curve (N) displays means in 20 healthy subjects.

to severely impaired. In one patient (with tumour of the cerebello-pontine angle) tracking was completely abolished in the direction towards the lesion, but was within normal limits in the opposite direction. The whole group exhibited a significantly reduced maximum velocity gain of smooth pursuit (Fig. 3). The difference was statistically significant ($p < 0.01$) at target velocities of 30°s^{-1} and above. The total amplitude of tracking was reduced at every target velocity (Fig. 3). The subjects also exhibited a decrease in the amplitude of smooth pursuit. The superimposed saccades with amplitudes of $3-10^\circ$, as well as of $11-20^\circ$ (Figs. 3 and 5), occurred more frequently than in normal subjects ($p < 0.05$ in both amplitude classes). The number of square waves was not significantly different from the normal values (Fig. 6).

Brain stem disorders

The ability to track was in general severely impaired in this group of patients. The maximum velocity gain of smooth pursuit was strongly reduced (Fig. 4). The difference was statistically significant ($p < 0.001$) at all velocities. The total amplitude of tracking was more reduced than in cerebellar patients but the amplitude of smooth pursuit was, in fact, reduced to the same extent as in the cerebellar group. The frequency of superimposed saccades was increased (Fig. 4). The difference was statistically significant only in saccades in amplitude class of $3-10^\circ$ ($p < 0.05$), while saccades larger than 10° and square waves occurred with the same frequency as in normal subjects (Figs. 5 and 6).

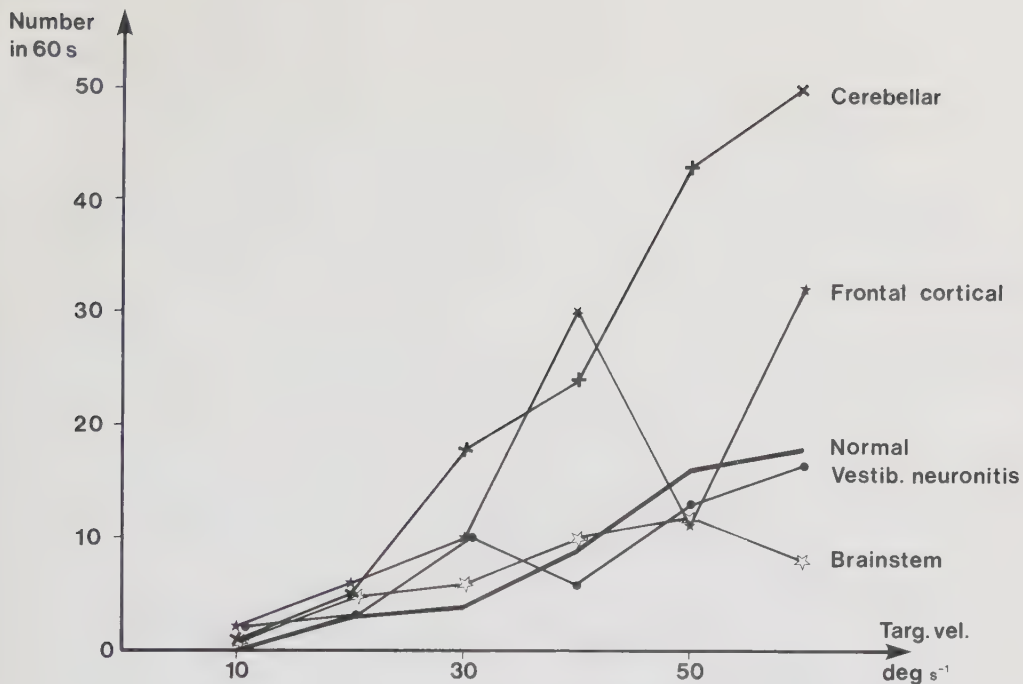


Fig. 5. Frequency of superimposed saccades of amplitude range of 11-20° during 60 sec of tracking. Mean number at six target velocities in the four groups of patients with different types of neurological disorders and in normal subjects.

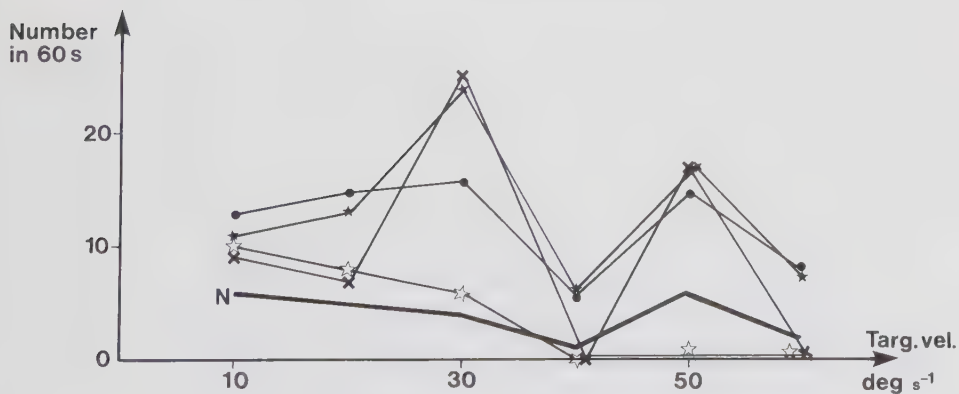


Fig. 6. Frequency of square waves during 60 sec of tracking at six target velocities. Mean number in normal subjects (N), in patients with vestibular neuronitis (●), with disorders of the frontal cerebral cortex (★), cerebellum (X), and brain stem (☆).

DISCUSSION

The disordered tracking exhibited following pattern: decreased maximum velocity gain and amplitude of smooth pursuit, followed by increase in frequency of superimposed saccades of $3-10^{\circ}$ in combination with decreased or normal total amplitude of tracking. However, the analysis of the four groups of patients revealed a gradation of impairment related to the type of disorder. Thus, the parameters studied were only slightly affected in vestibular neuronitis, moderately in cortical frontal disorders and severely in cerebellar and brain stem disorders. Moreover, the vestibular neuronitis and cerebellar groups showed features different from the other groups. Thus, the pattern in the vestibular neuronitis group was: an increase in superimposed saccades of $3-10^{\circ}$ in the direction of the fast phase of spontaneous nystagmus, in combination with otherwise normal parameters. The group with cerebellar disorders showed the reduced maximum velocity gain and amplitude of smooth pursuit which were combined with abnormally frequent saccades of both low amplitude (range $3-10^{\circ}$) and of large amplitude (range $11-20^{\circ}$).

The discussion will now turn to the possible importance of different structures within the nervous system (vestibular peripheral organ, frontal cerebral cortex, cerebellum and brain stem) necessary for the control of eye movements during tracking. Clinical aspects of the tracking test will also be discussed.

Peripheral vestibular imbalance - disturbing release of saccades during the tracking?

Peripheral vestibular disorder has been found neither to affect the execution of ocular tracking (Sakata & Umeda, 1976) nor to influence permanently the correct matching of eye velocity with target velocity (Baloh et al., 1977). In our patients with vestibular neuronitis, the normal maximum velocity gain of smooth pur-

suit and total tracking amplitude indicated that the control functions of the eye motor system in those patients were well preserved. However, reduction in amplitude of smooth pursuit to about 75% of the normal value gave us reason to assume that the vestibular imbalance in those patients might have caused deterioration in visual control more often than in normal subjects.

The significant increase in superimposed saccades only in direction concordant with the fast phase of spontaneous nystagmus suggests that the saccades appearing were not exclusively secondary to fluctuations in smooth pursuit, but might have resulted from a disorder of saccade release. The mechanism of this phenomenon is not clear and further analysis is called for in order to elucidate the finding.

Is frontal cortex essential for adjustment of tracking?

As found by Baloh et al. (1977) impairment of velocity of smooth pursuit in patients with fronto-parietal tumours might indicate the importance of the cortex for control of ocular tracking. However, in a recent report on eye movement patterns in patients with dyspraxia of speech (Dahlen et al., 1980), rather varying results of pendulum tracking - from normal to severely impaired - were described. In addition, quantification of tracking at different velocities of the stimulus in the present material also shows a considerable variability in the individual results. In the present study the maximum velocity gain of smooth pursuit was significantly reduced for the whole group, but only at target velocities above the optimal limits for smooth pursuit (40°s^{-1}). Thus, the frontal cortex did not appear crucial for modification of the velocity of smooth pursuit. We would favour the theory that the frontal cortex (Bizzi & Schiller, 1970), like the posterior parietal cortex (Lynch et al., 1977; Hyvärinen, 1978) are both involved in monitoring the

information on the movement.

Whether the increased saccadition during tracking in this group of patients was due to a decrease in maximum velocity gain of smooth pursuit, or due to poor cortical inhibition of superimposed saccades (Lynch et al., 1977; Dahlen et al., 1980) could not be concluded from the present results.

The cerebellum and the brain stem in control of smooth pursuit

In our patients, neither the mean values of maximum velocity gain of smooth pursuit nor its amplitude differed in patients with cerebellar and brain stem disorders. Lesions originating in one of those anatomic sites could have impinged on the other. As with other authors investigating patients with ponto-cerebellar disorders (Dichgans & Jung, 1975; Zee et al., 1976), we were unable to define the exact limitations of the disorder in our clinical subjects. Reduction of maximum velocity gain and of amplitude of smooth pursuit was more pronounced in patients with cerebellar and brain stem disorders than in patients with disturbances in the frontal cortex. This finding supports the concept that pathways and structures in posterior fossa are of great importance in visually mediated oculomotor control (Ito et al., 1973; Maekawa & Simpson, 1973; Takemori & Cohen, 1974; Precht, 1975).

In the brain stem group the maximum velocity gain of smooth pursuit was strongly reduced at all target velocities, while the cerebellar disorder group exhibited a significant reduction in the maximum velocity gain only at target velocities of 30°s^{-1} or above. This finding accords with a theory that execution of tracking eye movements is limited to brain stem structures (Raphan & Cohen, 1978), while the cerebellum utilizes the visual velocity information (Ito et al., 1973) for regulating the activity of pontine neurons

(Robinson, 1976).

Amplitude of superimposed saccades - an important parameter in posterior fossa lesions

When the tracking is performed beyond the optimal velocity limits for continuous feedback (40°s^{-1}), saccades with amplitude greater than 10° appear more often than when tracking slowly moving targets (Schalén, 1980) or during resting gaze (Bahill et al., 1975). It is conceivable that the saccadic system is activated to a greater extent in such conditions.

Hartje & Kerschensteiner (1973) pointed out that the amplitude of superimposed saccades was of no interest from a diagnostic point of view. However, they used only one target velocity (sinusoidal target movement of 30° amplitude, frequency 0.6 Hz corresponding to 18°s^{-1}) and their grounds for selecting clinical subjects (patients with unilateral cerebral hemispheric lesion) were different from those of the present study.

Our results suggest that the frequency of superimposed saccades with amplitudes of $11\text{--}20^{\circ}$ is significantly higher in patients with cerebellar disorders than in normal subjects at target velocities above 50°s^{-1} . Because a similar increase in frequency of larger saccades was not observed in any other patient group in the present material, it is possible that an increase in the number of superimposed saccades of large amplitude might be related to cerebellar dysfunction.

Clinical implications of the tracking test and its limitations

Like other precision movements, the tracking eye movements become deranged in neurological diseases. An altered pattern of tracking eye movement can therefore be an indicator of an underlying neurological disorder (Jung & Kornhuber, 1964). However, psycho-

logical engagement during the test (Holzman et al., 1976), aging (Sharpe et al., 1979), and medication (Rashbass & Russell, 1961) are also factors which can influence tracking. Standardized tests and quantitative analysis of results will minimize sources of misinterpretation. Nevertheless, several problems can arise when evaluating results obtained in clinical subjects. Many disorders are heterogeneous; the extension of pathological processes varies; ongoing healing, compensation, and degeneration occur. Any of these factors can explain the pronounced variation in the eye motor disturbances found when comparing different subjects with the same clinical diagnosis.

Accordingly, in clinical work the tracking test alone cannot be used for evaluation of the diagnosis. However, quantification of the impairment of different tracking parameters may indicate a possible site of the lesion. It would therefore seem that the tracking test may prove a valuable supplementary tool in otoneurologic examination.

ZUSAMMENFASSUNG

Die Folgebewegungen des Auges (tracking eye movements) wurden an zwanzig gesunden Individuen sowie an vierundzwanzig Patienten registriert, die teils an einer Vestibularisneuritis, teils an einer Störung im Gebiet des frontalen Kortex, des Zerebellums oder des Hirnstammes erkrankt waren. Die Tests wurden mit sechs verschiedenen Winkelgeschwindigkeiten des Fixationslichtes ($10-60^{\circ}\text{s}^{-1}$) durchgeführt und anhand von vier Parametern analysiert. Die maximal erreichbare Winkelgeschwindigkeit der kontinuierlichen Folgebewegung (smooth pursuit) war normal in der Gruppe der Vestibularisneuritis, jedoch reduziert in den Gruppen mit einer Störung des frontalen Kortex, Zerebellums und des Hirnstammes. Bei der Analyse der Amplituden der 'smooth pursuit'-Bewegung fanden sich normale Werte in der Gruppe der Vestibularisneuritis und der Gruppe mit frontaler Läsion des Kortex. Niedrigere Werte ergab die Analyse in den Patientengruppen mit Störungen des Zerebellums und des Hirnstammes. Die Frequenz aufgelagerter Sakkaden im Amplitudenbereich von $3-10^{\circ}$ war signifikant erhöht bei allen Patientengruppen. Die an einer Vestibularisneuritis erkrankten Patienten zeigten allerdings nur eine Frequenzsteigerung während der Folgebewegung des Auges in Richtung des Spontan-nystagmus. Eine Frequenzsteigerung der aufgelagerten Sakkaden im Amplitudenbereich von $11-20^{\circ}$ fand man in der Patientengruppe mit zerebellären Störungen. Das Auftreten rektangulärer Wellen (square waves) war in keiner der Gruppen signifikant erhöht.

Die quantitative Analyse der Folgebewegungen des Auges kann somit wertvolle Hinweise bei der Lokalisation zentralnervöser Störungen geben.

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